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POSTOPERATIVE STATES OF EXCITEMENT

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Acute psychoses following operation were probably known to Ambroise Paré (sixteenth century), but the first good description of such a condition is attributed to Dupuytren (1819),[1] who published a report of a case of "delirium nervosum" (to be referred to later). The subject received increasing attention with the widespread use of antiseptics, and the writings of the middle of the century speak of the etiologic connection of these agents, especially iodoform, with the psychotic developments. After the introduction of asepsis these toxic manifestations became less frequent, but then interest centered on the anesthetics. Psychoses following anesthesia with chloroform, ether and nitrous oxide were described. On the whole, the belief that the psychoses owed their origin to the anesthetic was not accepted, and stress came to be laid on two major factors: (1) The preexistence of mental disorder, especially chronic alcoholism, which came to overt attention after operation. That is, factors not related directly to the operation were involved. Dupuytren's "delirium nervosum" is thought to be delirium tremens which was noticed only after operation. (2) The presence of factors arising more directly from the operative situation: sepsis, fever, dehydration, anemia, cachexia, overmedication and gastrointestinal stasis. This group gave rise to the acute deliria, transient or more prolonged, coming on soon after operation and characterized by disorientation, confusion, defective memory, delusions of fear and hallucinations.

In the late nineties, however, one sees the recognition in French writings (Picqué,[2] Le Dentu[3]) of psychoses with an origin less clearly

From the Henry Phipps Psychiatric Clinic, the Johns Hopkins Hospital.

1. Dupuytren, quoted by Pilcz, A.: Ueber postoperatives Irresein, Wien. klin. Wchnschr. **15**:908, 1902 (excellent summary of early work).

2. Picqué, L.: Du délire psychique post-opératoire, Ann. méd.-psychol. **8**:91, 1898.

3. Le Dentu: Sur un travail de M. Lucien Picqué intitulé: Les psychoses chirurgicales d'origine infectieuse; la stupeur primitives des opérés, Bull. Acad. de méd., Paris **66**:245, 1911.

traced to the operative situation, at least as far as toxic factors are concerned. These factors were remarkably slight if present at all. A number of varieties were described, coming on somewhat later than the previous group and running a subacute or chronic course.

Picqué drew attention to the necessity of discovering before operation what patients would be likely to cause trouble after operation through the play of certain difficulties of personality and psychotic development. Later (1911) he seems to have come to the conclusion that postoperative psychosis never occurred without some infection. This conclusion was vigorously contested by his colleague, Le Dentu,[3] who also believed that too great an insistence on the "constitutional" features, in many cases well disguised, was begging the question.

At that time psychiatric thinking was dominated by Magnan's ideas concerning degeneracy; therefore it is not surprising that the unfortunate victim of this sort of illness was thought to be heavily predisposed constitutionally to the disease and that the operation was regarded merely as the precipitating factor. As things stood, therefore, if the patient became psychotic through infection, drugs, cachexia or other agents the operation, per se, would not be blamed. If he became psychotic without the significant influence of these factors, the blame fell on his constitution. Much quibbling ensued over the relative influence of toxic (infectious) factors and hereditary and constitutional predisposition, and in the argument the plastic facts of the situation itself and the patient's reaction to it were largely ignored. This was perhaps excusable at the time, as in the general thinking psychogenic factors were submerged as having no scientific value, and perhaps also because of the great urge to give the rapidly expanding surgical science a clean bill of health.

A noteworthy exception to this curious blindness to the obvious facts of the psychogenic factors of setting, the patient's attitude to the operation and other factors is seen in the literature on the psychoses following extraction of a cataract, which early attracted attention. Rudolf Löwy,[4] among others, called attention to the suspiciousness, panic-like excitement and motor overactivity, with fear and insecurity, arising in such patients generally at night, through the loss of normal landmarks. Any one who has seen such cases will recognize the essential completeness of Löwy's description and understanding of the cases.

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4. Löwy, R.: Geistesstörung nach Cataract Extraction, Allg. Ztschr. f. Psychiat. **52**:166, 1896.

I saw a case similar in all respects to the psychosis following extraction of a cataract in a young girl with an exophthalmic goiter, who underwent an operation for suturing of the eyelids on one side and their approximation with adhesive tape on the other side for the treatment of corneal ulcers (this followed some time after the thyroidectomy, with no unfavorable sequelae). She awoke in the early morning hours, became excited, fearful and suspicious of the environment; she heard her mother talking in the room and her physicians threatening her (auditory hallucinations). The excitement lasted about twelve hours and was terminated by a large dose of a derivative of barbituric acid. There were no recurrences or sequelae. When one remembers the great dependence on the eyes for ordinary orientation and the great faith with which most patients with ocular troubles involving an impairment of vision submit to an operation, it is easy to understand the dismay which they must feel when, after a few hours of sleep, they waken to find themselves in absolute darkness and stillness, with no one near to talk with. In such cases it seems reasonable to tax the psychogenic rather than the constitutional and exogenic factors for the bulk of the trouble.

Bonhoeffer [5] brought order into the psychoses associated with intoxications and infections by his classification of symptomatic psychoses. He recognized the great diversity of etiologic factors involved in the production of the symptomatic psychoses.

Kleist [6] added the postoperative psychoses to Bonhoeffer's group of symptomatic psychoses and listed as varieties the benumbing reactions with sudden outbursts, delirious states, anxious excitement, stupor, hyperkinetic excitement and paranoid states. They have in common a disturbance in orientation, difficulty in thinking and in focusing attention, recent deficiency of memory and amnesias. Kleist's group referred specifically to cases in which infection, disturbances of internal secretion, intoxication produced by drugs and other such obvious causes played no important rôle. He stressed postoperative malnutrition and dehydration and the absorption of toxic substances from the site of operation. He also stressed a constitutional predisposition through "psychopathic anlage, preexistent mental disease and senile brain changes" in one half of his cases.

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5. Bonhoeffer: Die Psychosen im Gefolge von akuten Infectionen, Allgemeinerkrankungen und inneren Erkrankungen, in Aschaffenburg, G.: Handbuch der Psychiatrie, Leipzig, Franz Deuticke, 1912, pt. 3, no. 1.

6. Kleist, K.: Postoperative Psychosen, Berlin, Julius Springer, 1916.

Among the varieties of symptomatic psychoses is the "amentia" picture, described by Bonhoeffer:[7]

> . . . outspoken thinking difficulty in the nature of incoherence, in the presence of a certain degree of benumbing . . . orientation in time or space is disturbed or absent. . . . Tendency to illusion, forced registering of sensory impression, occasional associations through rhyming and assonance, sensory illusions, especially visual and auditory, sometimes as scenery or dreams of delirium, sometimes circumscribed in a more distinct form of single sensory impressions, disturbing the stream of thought. Incoherent delusions often appearing of primitive origin, often in the sense of anxiety, of prejudice, of grandeur, quite phantastic and absurd, a recapitulation of indifferent reminiscences is observed.

The mood changes with the stream of thought (content) and is entirely congruous. Episodes of push of talk without other evidence of hyperkinesis are noted. More formal, catatonic-like disturbances in speech and motility are seen. A single case may show a predominance of one feature; Bonhoeffer considers this as of no special significance. He considered the syndrome of amentia to be always dependent on infection and so allied it with the symptomatic psychoses. In this respect he broke with his predecessors, who considered amentia a disease sui generis. Curiously enough, Bonhoeffer believed with Strohmayer that the cases of amentia based on mild intoxications were primarily hereditarily determined, and he placed no importance on such situational factors as sorrow, wrongs and strain.

Bonhoeffer wrote, relative to the etiology of amentia:

> In practice most cases of amentia are based on infectious diseases. . . .
>
> One point which still needs clarification is the significance of the psychogenic factors. Hoche considered them of equal importance with the physical factors. My own experience does not allow me this view, and I rather believe that a careful catamnestic revision of the cases in which only psychogenic factors of an emotional sort allow the demonstration will often show that other psychoses have been present. Episodic excitements of the degenerate, hysterical, epileptiform acute type, catatonia and others may occur. Certainly in many cases sorrow and worries about business can act as precipitating factors, especially when these factors are persistent and when inadequate nutrition and insufficient sleep are added. But the psychic etiology then works secondarily through the somatic changes, which like anemic cachectic states work as toxic processes. . . . That emotional precipitating factors can give to the outbreak of the psychosis . . . the last push, without therefore being the real cause, may be mentioned. I do

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7. Bonhoeffer,[5] pp. 66 and 71. Stransky, E.: Lehrbuch der allgemeinen und speziellen Psychiatrie, Leipzig, F. C. W. Vogel, 1919, vol. 2, p. 288.

not differ from Stransky in the belief that in the great majority of such cases the final cause arises from toxic factors.

In this country the psychiatrists Rohé [8] and Hurd [9] discussed the subject. Rohé stressed the factor of infection, and urged surgeons not to depend exclusively on the thermometer for its detection. Hurd stressed the constitutional factors and urged a thorough study of the personality before operation. These two works of psychiatrists stand in strange contrast to the later American contributions from surgeons. It is strange to see psychiatrists urging consideration of the "physical" factors and surgeons of the "mental!"

Following Rohé and Hurd, two of the most useful contributions to the subject of postoperative psychoses were made by surgeons, Kelly [10] and DaCosta.[11] Kelly saw 40 cases in 1,600 operations. He found them to be most frequent in children, in the aged, and in women between the ages of 35 and 45. He considered that anesthesia, physical shock and the kind and severity of the operation were unimportant causative agents. Aside from infections, auto-intoxications and intoxication from drugs, he considered an "unstable nervous system," and especially anxiety and worry about the operation and the trouble which led to it, the most important factors. He stressed prophylaxis through the quieting reassurance of good nursing.

DaCosta agreed with Kelly in all essentials (even regarding the incidence of the trouble), and quite rightly linked postoperative with puerperal psychoses. DaCosta stated:

> The patient may have long suffered from fear, pain, insomnia, or exhaustion; he worries before, and probably takes a general anesthetic at the time of the operation. The operation produces shock and loss of blood and the anesthetic may poison him. After the operation there may be postoperative pain, insomnia, anxiety, fear and homesickness. . . . In all cases of postoperative insanity there is predisposition, hereditary or acquired. . . . A normal, stable, healthy brain will probably never go insane after an operation unless that operation

8. Rohé, George H.: Postoperative Insanity, Am. J. Obst. & Gynec. **39**:324, 1899.

9. Hurd, Henry M.: Undetected Tendencies to Mental Disease, Am. J. Obst. & Gynec. **39**:331, 1899.

10. Kelly, H. A.: Postoperative Psychoses, Am. J. Obst. & Gynec. **59**:1035, 1909.

11. DaCosta, J. C.: Diagnosis of Postoperative Insanity, Surg., Gynec. & Obst. **11**:577, 1910.

attacked the brain, removed the testicles, or removed the ovaries. . . . The two most active causal elements of insanity associated with surgical operation are fear and worry. . . . Postoperative insanity is practically identical in nature and mode of genesis with puerperal insanity.

Considering the exclusive stress which continental writings laid on infection and predisposition, the conclusions of these two American surgeons are striking in their psychobiologic inclusiveness. One may not be too harsh with DaCosta because of the synonymous use of "brain" with mental function, or with his stressing of predisposition. Much can be forgiven because of the proper emphasis placed on the psychogenic factors.

I believe that the cause of understanding of psychoses is poorly served by too great an insistence on nosologic distinctions. If one grants that any case may and should be studied for the contribution to it of exogenic, organogenic, neurogenic, psychogenic and constitutional elements, a fairer diagnostic appraisal will very likely ensue. I therefore hesitate to ascribe to certain factors a "precipitating" influence, while the true "etiologic" influence is allotted to others. According to Adolf Meyer, a "fact" is anything which can become a "factor" in the case. Who can say therefore with any degree of certainty what is primary and what secondary? Historically, at least, the psychogenic elements in postoperative psychoses have been minimized, and this has been done at times with the appearance of attempting to justify surgical interventions and to whitewash situational at the expense of "constitutional" factors.

REPORTS OF CASES

The four cases to be reported illustrate an unusual type of excitement seen after operation and show the difficulties encountered in assaying a reaction for its component etiologic elements: exogenic (toxic-infectious-metabolic), neurogenic, organogenic, psychogenic (situation-experience determined) and constitutional (personality-hereditary). They also shed light on the problem of the so-called postoperative psychoses and perhaps help in their reevaluation.

CASE 1.—A married woman, aged 32, complained of sterility and of dysmenorrhea of two years' duration. An operation was advised, and she agreed without hesitation. She was admitted to the hospital on Jan. 25, 1933, and on January 27 a Baldy-Webster suspension of the uterus and an appendectomy were performed under nitrous oxide and ether anesthesia. The operation was uneventful, except that the small intestine was nicked and repaired. The condition was

good immediately following the operation. The postoperative course, determined from the hospital record, nurses' notes and the information from the family, was:

Postoperative Day	Physical Condition	Treatment	Data on Behavior
1	Weak, nauseated	Hypodermics of morphine ⅙ grain (0.01 Gm.) every four hours	Resentful of hypodermics, temper tantrums; had husband discharge two nurses for slamming doors, scaring her
2	Felt weaker	Hypodermics of morphine ⅙ grain every four nours	Cried; nervous and uneasy over hypodermics; upset because relatives were allowed to remain only a few minutes
3	Complained of abdominal cramps, believed to be menstrual; itching macular rash on palms, legs, mouth (probably due to bromide)		
4	Condition good		
5	Temperature practically normal; rash and cramps disappearing		
6	Slight fever		
7	.....	.....	Visited by nurse she had discharged, who slammed door again; patient uneasy; began to smell "sweet gas" (later referred to as hospital odor)
8	.....	.....	Same nurse visited her with hypnotic medication; patient refused pills, but took milk; wakened in night, "numb all over," and frightened by the experience; fell asleep again
9	.....	.....	Felt cold; physician on rounds remarked she had "taken a turn for the worse"; her family then began rubbing her extremities to warm her; patient fearful of dying, felt her pulse, announced she would die if husband did not come before 3 p. m. Began counting 1, 2, 3..1, 2, 3..4, 5, 6..4, 5, 6 a, b, c..a, b, c, etc.; repeated verbatim questions asked by visitors
10	Fever; refused food and fluids	.....	Excited; restrained by being tied down; excitement worse thereon; resentful of restraint
11	Blood pressure, 96 systolic and 60 diastolic after intravenous treatment	Sodium amytal 5¾ grains (0.248 Gm.) intravenously 11:30 p. m.	Very restless; (physician's notes "hysteria")
12	No note	No note	No note
13	No note	No note	Very profane; cursed nurses
14	No note	No note	No note
15	....	.....	Seemed normal; oriented; asked husband to have her watch repaired; did not know how she had broken it
16	No note	No note	
18	No note	No note	No note
19	.....	.....	Noisy, talking in a high-pitched voice; seemed to know where she was; talked of nurse and physician; "flight"; no delusions or hallucinations
20	....	.....	Fearful, suspicious, resentful, excited; restrained by being tied hand and foot and having heavy canvas sheet put over her
2/16/33.....		Psychiatric consultation	Disoriented for time
		Transferred to Henry Phipps Psychiatric Clinic	

The patient was transferred in an ambulance. She was excited, overactive and fearful. The husband said that she believed the physicians at the other hospital were against her and had put a spring or wire in the operative site; she believed that the nurses were making signs behind her back and that the food contained drugs.

Physical Examination.—On admission the temperature was 103 F. (rectally), the pulse rate from 124 to 152 and the respirations from 24 to 26. The skin was dry, with a rash over the buttocks, thighs and back. There were a few palpable lymph nodes at the base of the neck. Dermographia was present. The lips were cracked and parched; the breath was acrid. The abdominal scar was in good condition.

Laboratory Examination: The hemoglobin was 14.4 Gm.; the white blood cells, 11,100, with 80 per cent polymorphonuclear neutrophils; the sedimentation rate, 41 mm. per hour. The urine contained a trace of albumin. The blood bromide was 125 mg. per hundred cubic centimeters.

The patient was oriented for time, place and person. She was so excited that she was placed in a cold wet pack, which only increased the excitement and fear. She was removed immediately. She made pelvic movements of coitus when the husband said goodbye to her.

Course in the Clinic.—The patient continued excited, with push of talk (see later), making flirtatious gestures to the physicians with the appearance of elation. The excitement consisted mainly of rhythmic movements of the arms and feet, in time with the stream of talk. She never tried to get out of bed. She remained resentful of questioning by the physicians but talked with the nurses. She made demands.

The stream of talk was measured, in nursery-rhyme style, slow enough to be taken down in longhand by the nurses; it was often accompanied by rhythmic movements of the extremities.

The following is a verbatim example obtained on the day after admission to the clinic:

"Close the door—please. They are coming to kill me. If you say she—they will say me! Tom—oh, Tom. They have killed you twice—I am not trying to kill you; I am not trying to kill you. You do love your mother. I haven't one now. She has gone. She was in heaven. Now she is dead. She has gone to rest—rest—just rest—and rest. Mother's gone and she is dead. Don't kill her. Don't kill her. Don't kill my mother. She was in heaven. She is in heaven. Kill Tom! He didn't have a mother—and I don't either. I have a father. (Is he living?) My father is dead. He is in heaven. He is on the cross. Please don't tell. I mean don't kill us. Don't kill us all. They will—they will. Don't kill—please don't kill. Tom—my Tom! She knows she is dead. Please don't shoot!—me—don't shoot me twice. I have got to say—three or four more words—please, oh, please. When they are across—they say all right. Oh. please—oh, please—don't send us across—across the great divide. I know your mother is dead. Oh, don't say it! Don't kill us twice. Please don't say—Tom—I know your mother is dead—because she is dead—and he has no mother too. Now you are going to kill—going to send us all through—aren't you? You know—and I know—it is a great world. If you do—go to hell—say all right—then go straight to hell. Please—oh, please—don't—don't! Don't send us some more—go straight to hell. . . . I have a big brother—but I am a little sister. He died to save me from hell—and he died—and I died too. I have got a big brother—on the other side. He died all right—and he left me die—and I died too. Well then I will live

—well then he won't live—because I really have a big brother dead—and a little sister too—and if I live—he will live too—but if she is wrong—I know I will say—I will say *she—she.* I shouldn't say she—she—you live for me—I will say big brother—live for me—you live for me. I have a big brother—and he died too. I won't say yes—yes—I won't—but I will say it wrong. He is living and I am wrong—because I am on the great other side—and you know—and I know—I am wrong—and if I do—you know where I will go. I will go to heaven—and I will go to hell. I have got a big brother dead—on the other side—and if I die—won't you shoot me—shoot right when I say she—she. I will say she—she—and she will too. I have a big brother too—send us all into hell. I have a big sister—and she does too. Can't you see—see—she died—and I died—and he died and she died too. You will say—she—she—she—she means that he is dead. Is it cold? I am dead—it isn't cold—please, no. I don't mean that—it means my brother is dead—and your brother is dead. If you want to shoot me—say me—me. Me—me—she—she—she! She—she—she—she knows where I am. She will say—she—she—if you say—she—she! . . ."

From this sample several features of interest emerge: (1) push of talk; (2) the rather repetitious character, without the free unrestrained flight of the manic excitement; (3) definite topics with a fearful import; (4) the question-answer construction; (5) a certain progression, by contrasts or other easy association; (6) a lack of distractibility.

The patient remained in this condition for about ten days, then she gradually began to improve. Sleep and appetite were better. The pulse rate fell. The temperature returned to normal, together with the white cell and the differential count; there was better intake of fluid and food. Only the sedimentation rate remained elevated (on February 23 it was 45 mm.). On February 27 there was a short-lived rise of temperature to 103.4 F., with a white cell count of 14,800, and 80 per cent polymorphonuclear neutrophils. The patient was allowed to get up gradually, and there developed a persistent mild rise in temperature to 100 F. in the afternoon, with a persistently high sedimentation rate (on March 22 it was 52 mm.; on April 6, 43 mm.). There was a painless mass in the culdesac; this persisted but became slightly smaller as time went on. This recalled the injury to the small intestine at operation. Other search for foci of infection gave entirely negative results.

The patient began to show temper tantrums with the improvement. She said that she had always been spoiled by her husband and had utilized these displays whenever necessary to get her way. On the whole she proved tractable, showed initiative and considerable social poise, and was a good influence with other patients.

At this stage she interpreted some of the content as follows: The sweet gas she had smelled in the hospital was referred to as a hospital odor. The ambulance was a hearse, and she was on her way to the cemetery at the time of her transfer to the clinic.

She was discharged on April 6 as well. The only abnormal finding was the sedimentation rate of 43 mm. per hour. She has remained well.

A review of the patient's past brought little additional material of significance in the case. There had been some doubt as to the husband's fidelity a few months before, a quarrel and a reconciliation. She was anxious to become pregnant. She evaded discussion of these factors.

Summary.—There were mild delirium ("amentia") from dehydration, a postoperative state and the excessive use of drugs (opiates and

bromides), in a setting of fear and misunderstanding of postoperative treatment and irritation by hospital personnel, together with growing resentment, excitement and elation. The latter features were rhythmic talk of a repetitious, topical nature, delivered in a sing-song voice, expressing fear but without the appearance of fear. Gradual subsidence of the excitement occurred with improvement in the physical condition. Relatively little amnesia remained. The fearful content came to a climax with the transfer to the psychiatric hospital by ambulance, which was interpreted as a hearse. The persecutory delusions and the olfactory misinterpretation are explicable on the basis of the toxic condition and the feeling of being at a disadvantage in the environment.

In this case several factors were at work: Toxic factors consisted of: operative intervention (absorption of toxic products), dehydration, undernourishment, fever, probable focus of infection in the culdesac and bromides, and opiates. The neurogenic and organogenic factors were not determined (a lumbar puncture was not done). Psychogenic factors were: poor after-treatment with excessive and ill advised hypodermics, slamming of doors (perhaps only interpreted as such by the patient in an apprehensive state of tension) and revisiting by a nurse she disliked. These factors bred an uncooperative attitude, leading to difficulties in maintaining a proper fluid and food balance. A feeling of insecurity with the husband, and a motive in desiring a child after ten years of married life and soon after the quarrel with the husband over his infidelity remained as more remote factors. Constitutional features were temper tantrums and the fact that the patient was spoiled.

There was no evidence of any nervous or mental disease in the family. The patient had never been ill before.

As is usual with deliria, there was little subsequent effort to collaborate in the investigation of the psychogenic data. To her the operation offered a sufficient cause for the development of the trouble, and there developed no curiosity in regard to the contribution personality made to the event. The constitutional features offered certain minor difficulties in the practical management of the case.

CASE 2.—A man, aged 40, married, a farmer, who was admitted to the Henry Phipps Psychiatric Clinic on May 15, 1933, had suffered for months from pain in the right sciatic region and had seen several physicians for the trouble. Finally the condition was called appendicitis, and the patient was referred to a surgeon in a nearby city for operation. After examination the surgeon could not confirm the diagnosis and favored an extraction of some teeth and a tonsillectomy. There was some intention to operate on the sinuses later.

The patient was admitted to the local hospital on May 3, 1933. He dreaded hospitals and always said, "You might as well wheel me to the cemetery." On May 3, 1933, he submitted to a tonsillectomy, the extraction of five teeth and the removal of a nasal polyp. He was apprehensive before the operation and struggled during the anesthesia.

The chronological order of events follows:

Postoperative Day	Physical Condition	Treatment	Data on Behavior
1	Constipated; could not use the left arm; this was thought to be due to lying on it during the operation; "no special temperature"	.....	Restless
2	Constipated	.....	Restless; sleepless; felt the bed going round
3	Constipated	.....	Restless; sleepless; felt the bed going round
4	Constipated	Morphine	Day: restless, called for "water, water, water"; wet bed; speech "confused," talk vague
		Sodium amytal 0.6 Gm. intravenously	Evening: Wildly excited, jumping out of bed, tearing bed clothes and those of fellow patients, shouting; believed people were against him; had to be restrained
5	Constipated; 1 bowel movement in six days, patient worried over this	Sodium amytal 1 Gm.	"Wild," spitting, kicking, crying out; transferred home under hypnotic medication at the request of the hospital as unmanageable there; later awoke, composed, and said he had lost control of himself
6	.....	.....	Sleepless; occasional periods of mild confusion; spirits good most of the time
7	.....	.....	Sleepless; occasional periods of mild confusion; spirits good most of the time; many visitors
8	.....	.....	Sleepless; occasionally mildly confused; concerned over the running of the farm
9	.....	.....	Sleepless; occasional periods of mild confusion
10	.....	.....	Sleepless; occasional periods of mild confusion
11	.....	.....	Excited, shouting, singing, whistling for his horses; spoke of having had a vision from above, of having seen and spoken to a long lost aunt; transferred to Henry Phipps Psychatric Clinic (a trip of 400 miles) by ambulance

The patient arrived at the clinic at 1 a. m. on May 15, 1933. He was in a very excited state, overactive, with great push of talk and some distractibility. He appeared suspicious and apprehensive, but at times he smiled and laughed aloud. Owing to the serious physical condition a complete examination of the mental status was not attempted until later in the day.

Physical Examination.—The patient was dehydrated; the axillary temperature was 102.6 F.; the pulse rate, 114; the respirations, 26; the weight, 119 pounds (54 Kg.). The blood pressure was 110 systolic and 78 diastolic. There were a mucopurulent nasal discharge and necrotic areas in the tonsillar fossae; the abdomen was tense and distended, with some tenderness in the right lower quadrant. There was sciatic tenderness on the right. The white cell count was 23,400, with 90 per cent polymorphonuclear neutrophils. There were numerous granular casts in the urine.

Course.—The patient continued to shout excitedly, uttered erotic talk and handled the genitalia, saying, "I'm all right down here." He appeared dis-

oriented, but examination of the sensorium was impossible. He was given scopolamine hydrobromide 1/100 grain (0.00065 Gm.) and morphine 1/4 grain (0.016 Gm.), which failed to quiet him. He collapsed in a cold wet pack and had to be removed. He then remained quiet and slept the rest of the night. Five hundred cubic centimeters of urine was removed by catheter and a solution of dextrose and sodium chloride was administered by Murphy drip.

When he awoke at 8 a. m. the patient was weak and quiet, lying peacefully in bed, but suspicious and distrustful in his attitude. He did not know where he was and asked for his relatives and for his physician. He refused food until relatives had reassured him. Later in the morning he again became restless and overtalkative; he talked in a low voice, accompanying the talk with rhythmic pronation-supination movements of the legs and beating time with the hands.

A verbatim sample of talk on May 15 follows: "Anything you say. Anything at all except the last thing. Being dead and covered up. Anything—anything, it's all right. Anything you please. Everything is all right. Bowels moving, teeth pulled—anything, it's all right. Anything but being dead and covered up. Anything you can do to keep me alive. Being dead and covered up. Do anything to save me. Anything. Anything you can do. That's all right, that's all right. Anything you like. That's all right, pull worms out of me. It's all right. Anything you can do—it's all right. I'll move my bowels—anything you ask me, to keep me alive. Being dead and covered up. Being dead and covered up. I'll do anything, anything but the last thing. Anything, anything—move my bowels, anything to save me. That's all right, anything you say. That's all right. (How are you today?) I am feeling good. I'll do anything you tell me to save me. Anything—it's all right. Anything you please—anything you say. It's all right, it's all right. Anything but being dead and covered up. Anything you can do to save me. I'll move my bowels—anything you say. That's all right (repeated many times). (How do you feel this morning?) I feel all right. I'm all right. (Last night?) All right. Everything is all right. (How long have you been here?) All my life. Just so you can keep me alive, everything is all right. I'm all right. . . . Ticking and tucking. It's all right. Everything is all right. That's all right. I am all right. Just so I ain't dead. I'll do anything. Just so I ain't dead. I'm all right. That's all right. Sticking and stuffing. It's all right. Just so I ain't dead. Sticking and stuffing. (Patient sees tube at foot of bed)—Cutting off that tube. That's all right. (Do you know this young lady?) Yes, she's a nurse. Just cut that tube. Ticking and stuffing. I'm all right. Ticking and stuffing. I'm all right. (Any bad dreams?) Yes, I thought I was dead. Everything's all right. Though they cut off this arm. (Did they?) No, they didn't cut that arm off—just took the worms out. (Did they operate on your throat?) No. Ticking and tucking—anything but being dead. Moving and sticking and stuffing and anything, so I am not dead. (Do you know where Baltimore is?) Yes, anything. Just sticking and stuffing, anything you say—I'll do anything. I just thought I was dead and covered up. Felt good this morning. Just cut that tube off. Pull the worms out of me. I'm all right. Just so I'm not dead. I'm all right. Everything is all right."

From this sample of stream of talk are seen: (1) push; (2) mild distractibility without flight; (3) repetitiousness, and (4) topical material, featuring fears and misapprehensions.

In this connection the patient said later (May 18) that he thought he was in a casket in a hearse when coming to the hospital by ambulance (driven by a local undertaker), and that he was going to be buried alive. His activity was designed to prove to the environment that he was not dead and so to prevent his burial.

When he arrived at the Henry Phipps Psychiatric Clinic he thought that he was in heaven and that the nurses were angels. On waking in the morning after admission and finding himself alive he was overjoyed, and felt that no matter what was done he would be all right. "Anything but the last thing" referred to death; "sticking and stuffing" to hypodermic injections; "cutting off the arm" to the numbness and partial palsy after operation (due to having lain on it). The numbness was demonstrable shortly after admission. "Worms" referred to a previous belief that he had worms, and he interpreted the insertion and removal of the rectal tube as the movement of the worms.

The patient became quiet again and seemed at ease, but in the evening he became restless and excited and fearful of being shot and operated on; he jumped out of bed, crawled underneath, kicked and yelled "Everything won't fit in the casket. If you go back she'll prove it." He slept after morphine and scopolamine hydrobromide had been administered hypodermically. The next day he was quiet but distrustful. Examination of the mental status showed that he was oriented for time and place but not for persons and that he possesed surprisingly good memory for both remote and recent events. After May 18 he remained pleasant, cheerful, oriented, cooperative and quiet. He gave a good retrospective account of the events of the illness, with his fears and elation at finding himself alive.

The physical condition improved remarkably with a high intake of fluids. The suspicion of an acute condition in the abdomen was not confirmed. The temperature reached normal on May 17 and remained normal. The pulse rate also became normal. The white cell count was 3,100, with 71 per cent polymorphonuclear neutrophils on May 16; the sedimentation rate was 42 mm. in one hour. The red cell count was 5,390,000 and the hemoglobin, 17.4 Gm. per hundred cubic centimeters on the same date. On May 17 the white cells numbered 10,500, with 80 per cent polymorphonuclear neutrophils; on May 19, 11,200, with 85 per cent polymorphonuclear neutrophils; on May 20, 20,000, with 81 per cent polymorphonuclear neutrophils; on May 24, 11,500, with 77 per cent polymorphonuclear neutrophils; on May 25, 8,400, with 66 per cent polymorphonuclear neutrophils, and a sedimentation rate of 51 mm. in one hour, and on June 5, 9,100, with 69 per cent polymorphonuclear neutrophils, and a sedimentation rate of 42 mm. in one hour.

On May 16, the blood chemistry showed: nonprotein nitrogen, 64 mg. and sugar, 137 mg. per hundred cubic centimeters; on May 17 the nonprotein nitrogen was 49 mg. and the sugar 111 mg. and on May 25, the nonprotein nitrogen was 34 mg., and the sugar, 92 mg.

The urine, which on admission had shown a few granular casts and a trace of albumin, was normal on May 26.

Roentgen examination of the sinuses on June 1 showed a chronic infection of the antrums and anterior ethmoids.

For the last two weeks of his stay in the hospital the patient was smiling, cheerful and showed no distrust or fear. He related a previous illness at the age of 23, soon after the birth of his first child. The flickering lights of a movie hurt his eyes and made him feel sick, and he had to leave the theater. On the next morning he was unable to speak and remained mute for about a week; then he recovered speech suddenly. He was weak, but not depressed, for several months thereafter.

There was nothing else of special significance in the past life. The patient was a simple man, leading a simple farming existence; he was happily married and had no special troubles. One sister was said to have had a "nervous spell" which did not require hospitalization.

The patient was discharged on June 2. Subsequent information obtained from him notes a gain in weight of 7 pounds (3.2 Kg.) in the first week at home, and a total gain of 23 pounds (10.4 Kg.) in three months. He has remained well except for a recurrence of the sciatica.

Summary.—A simple man, fearful of hospitals, underwent an operation which was not the one that his own physician had wanted. He struggled apprehensively under the anesthesia. Following the operation the left arm was numb and palsied, and he was constipated, restless, sleepless and dehydrated. Four days after the operation wild excitement occurred, which subsided temporarily after the patient was transferred home. The excitement recurred a week later, in a setting of sleeplessness, dehydration, constipation, fever, worry and fear. There were prodromal periods of mild confusion before each excitement. On being transferred to the Henry Phipps Psychiatric Clinic the patient believed that he was dead and about to be buried, and the reason for the wild excitement was to prove that he was not dead. On wakening he was elated to find himself still alive and became overtalkative. The physical status showed the toxic factors of fever, dehydration, infection in the operative site, a suspicious condition in the abdomen, polymorphonuclear leukocytosis, and a persistently high sedimentation rate.

In his past there had been a hysteria-like episode at the age of 23; this was not well defined, but was associated with mutism and weakness.

In this case there is more of the panic-like element, and the delirium is less clearly defined than in case 1. One is impressed, however, by the marked toxic features, and by the parallel improvement in the behavior and the physical condition. The term "amentia" seems to fit this case.

Transferring the patient home under the circumstances seems to have been a dubious procedure, especially since there seems to have been inadequate postoperative care available there. The case was rather remarkable for the completeness of the retrospective account of the fears and misinterpretations.

Case 3.—A woman, aged 43, married, who was admitted on March 23, 1933, had suffered from menorrhagia since October 1932. In November 1932, she had consulted a physician who told her that she had a tumor of the uterus, the size of an egg, and that it should be watched. She did not seem worried. In February 1933, the bleeding became more severe, and on March 1, 1933, she again consulted the physician. Following a vaginal examination she was told that a "scraping of the womb was absolutely necessary." Her behavior did not impress any one as abnormal, but later, in an unfinished letter, she referred to her spirits as being "low" at this time. The pulse rate was 56; the hemoglobin, 76 per cent, and the red blood cells, 4,010,000. Two tablets of a barbital derivative with amidopyrine were given at night. On March 2, the ordinary preoperative preparation was made, and on March 3 a dilatation and curettage, with complete hystero-salpingo-oophorectomy, were performed under avertin anesthesia (and ⅓ grain [0.021 Gm.] of a mixture of the hydrochlorides of the opium alkaloids). The patient experienced surgical shock after the operation.

The chronological order of events (from the physicians' and nurses' notes) follows:

Postoperative Day	Physical Condition	Treatment	Data on Behavior
1	Nausea and vomiting	Gastric lavage; infusion; mixture of the hydrochlorides of the opium alkaloids 4/3 grain (0.086 Gm.); codeine ½ grain (0.032 Gm.); acetylsalicylic acid 5 grains (0.32 Gm.)	Talk and behavior essentially normal; worried over the nausea and vomiting
2	Nausea and vomiting; gaseous eructation; flatus; pulse rate from 100 to 112	Gastric lavage; infusion; mixture of the hydrochlorides of the opium alkaloids 4/3 grain (0.086 Gm.); codeine ½ grain (0.032 Gm.); acetylsalicylic acid 10 grains (0.65 Gm.)	Very apprehensive
3	Vomited; abdomen distended; flatus; pulse rate from 104 to 126; hemoglobin 50 per cent; red blood cells 3,490,000; "condition poor"	Dextrose, 500 cc.; codeine ½ grain (0.032 Gm.)	Apprehensive
4	Temperature 100 F.; pulse rate, 128; respirations, 20	Blood transfusion at 2:45 a. m. without explanation; morphine 3/12 grain (0.016 Gm.); codeine ½ grain (0.032 Gm.); a barbital derivative with amidopyrine 1 tablet	Very apprehensive; spoke of Mayor Cermak having been given a blood transfusion as a last resort; later in day, told by her physician she had had a narrow escape from death
5	Urine: acetone 4+; taking fluids poorly	A barbital derivative with amidopyrine 2 tablets; codeine ½ grain (0.032 Gm.)	Told by her physician that the "organs had come out"; did not seem disturbed; had known before operation that this might be necessary; talked of dying; on the whole, less apprehensive; drowsy; taking fluids poorly
6	Temperature 100 F.	A barbital derivative with amidopyrine 2 tablets; 300 Gm. 10 per cent dextrose	More comfortable, refused food with carbohydrates
7	.....	A barbital derivative with amidopyrine 2 tablets	Talked and cried entire night; did not answer questions, appeared confused as to meanings
8	Temperature normal; sleeping poorly	A barbital derivative with amidopyrine 2 tablets; barbital (quantity ?)	Felt fine; no complaints; upset in evening
9	No note	No note	No note
10	No note	No note	No note
11	.....	Barbital (quantity ?)	Excellent spirits, writing poetry; talks much
12	.....	A barbital derivative with amidopyrine 2 tablets	Melancholic and having crying spell
13	.....	Dial 2 tablets; chloral hydrate 15 grains (0.97 Gm.)	Euphoric and talking incoherently
14	.....	Lumbar puncture; morphine ⅙ grain (0.01 Gm.); atropine 1/150 grain (0.4 Gm.)	Confused, euphoric and hysterical; talk of a new life and well-being
15	Cerebrospinal fluid: 3 cells; Kahn reaction negative; gold curve, mastic curve and globulin negative	.....	Refused food; disoriented with short periods of rational behavior
16	.....	Tube fed; hands bound down; a brand of phenobarbital U. S. P.; sodium amytal (quantity ?)	Agitated, tearing off clothes, talking continuously, with reiteration and perseveration of names of things seen or heard
17	No note	No note	No note
18	.....	Pentobarbital sodium, 1 tablet; sodium amytal 3 grains (0.195 Gm.); repeated if necessary	
19	Hemoglobin 66 per cent; red blood cells 3,190,000; white blood cells 7,550	Sodium amytal 6 grains (0.4 Gm.)	
20	Dehydrated; incision in good condition	Transferred by ambulance to Henry Phipps Psychiatric Clinic; sodium amytal 3 grains (0.195 Gm.)	

The patient was admitted in an overactive, excited state; she was distractible, showing push of talk and flight of ideas. The physical status showed dehydration, parched mucous membranes and coated tongue; the temperature was 99.4 F. (rectally); the pulse rate, 90; the respirations, from 20 to 22; the blood pressure, 180 systolic and 82 diastolic; there were some carious teeth. On the following day, additional examinations showed: nonprotein nitrogen, 42 mg., and sugar, 86 mg. per hundred cubic centimeters of blood, hemoglobin, 11.6 Gm., red blood cells, 4,040,000; white blood cells, 4,900, with 60 per cent polymorphonuclear neutrophils; the sedimentation rate was 53 mm. in one hour. There were no bromides in the blood. The Wassermann reaction of the blood was negative; the flocculation test was doubtful.

The stream of talk was as follows: "Look at the adhesive on my hip, it is gray—yes, battleships are gray. (Laughing hysterically—tearing gown. Pounding on wall.) I see a light—it's shining bright. Look—bars, a cell, a prison—oh—Irene Castle—yes, castles in Spain. I am making a fool of myself—oh my family. Mrs. Peterson, that reminds me. Pete—my little sister's husband. Oh captain, where are you captain. I can't see you. The Prince of Wales. Oh, if you would walk into my room now—(laughing, pulling hair). Irene Castle, oh Irene, where are you? Dr. B.'s wife, she died too—from cancer—no neuritis—no legs—why do I say legs? oh yes, Marlene Dietrich. Captain Byrd, where are you? Oh, I heard a shot (when water was turned on) a shot, oh, Mayor of Chicago—Mayor—he was shot, he was a friend of mine. Captain, why do I say captain, oh yes cap—Lupe Velez—The Cuban Love Song—Havana, Cuba, it is a beautiful place. Oh Irene Castle, Captain Emerson, where are you? I remember. Oh my boneless baby—that reminds me of the Bonus Marchers—I stood and watched them. Peter, he was great, no—that's Peter the Great. He was my third husband, oh did I have three husbands? (Put hands over mouth, laughing. Turning from side to side. When nurse came in room)—Oh, there you are, my day nurse, no you are Irene Castle. (Scratching head.) I'm bleeding, oh, captain, the pit, the pit of my stomach—there's a scar—that reminds me of a little crippled girl I saw. Oh, my God, there is a God, isn't there, Emerson—Marie Clemson—yes, Queen Marie, Russia, Balkan Sea—oh, keys, lock, a door (when door was opened). Lord—yes, he was a lord. Am I a cripple? I was afraid my baby would be a cripple, because my mother was a cripple.

I'm Eddie Cantor. Did I have a hemorrhage? Dr.—no, you're not the doctor, you're Eddie Cantor. Did my baby die; no, I died.

Didn't I have a baby, did it die—tell me—was it twins? I was bleeding to death—Oh, God, I was bleeding to death.

Did I have a cesarean section? Were they twins?

I had a bad nightmare, an awful dream, and it made me lose my mind.

Oh, tell me I'm alive, that I'm not dead.

Will you please tell me whether that man from Chicago died or is he better? They're keeping that news from me for some reason or another." Asked what the man's name was, she replied: "Mayor Cermak. They bled me at Johns Hopkins Hospital just like they bled him."

From this are noted: (1) push of talk; (2) distractibility, rhythm and flight of ideas; (3) topical material dealing with fear of dying, bleeding, delusional ideas of pregnancy, fear of physical disability in her child and ideas of insanity.

Course.—The patient remained in an excited manic state, with great verbal productiveness, elation and antagonism interspersed with more cooperative and lucid intervals. Her sleep was profoundly disturbed. The pulse rate ranged from

80 to 125, and the rectal temperature, from 98 to 101 F. Tube feeding was necessary. On May 8, the hemoglobin was 9.7 Gm.; the red blood cells, 4,280,000; the white blood cells, 8,600, with 67 per cent polymorphonuclear neutrophils, and the sedimentation rate, 38 mm. in the first hour.

The patient was transferred unimproved to another psychiatric hospital on June 10, and made a good recovery there, being discharged as cured on September 15.

The past history seemed to be noncontributory. The patient was happily married a second time, after leaving her first husband several years before because he infected her with syphilis. There was no evidence of syphilis, except the doubtfully positive flocculation test. There were no children. The patient had suffered no previous mental disturbance.

All informants stated that there had been no nervous and mental disease in the family, but during the excitement the patient stated that her mother died "in an insane asylum." This item of history was not checked later.

Summary.—A married woman, aged 43, moderately anemic from menorrhagia, submitted to operation for dilatation and curettage with the understanding of the possibility of more serious intervention. Dilatation and curettage and complete hystero-oophorectomy were performed under avertin anesthesia. Postoperative shock and much emergency treatment, including a blood transfusion, given early in the morning without an adequate explanation to the patient, gave rise to concern over her condition and to "low spirits" for eight days postoperatively. This was followed by fluctuation of spirits, with increasing toxicity evidenced in disorientation and confusion, and in marked anemia, an increased sedimentation rate, moderately high nonprotein nitrogen in the blood, mild fever and dehydration (the operative wound was in good condition). Physical improvement was accompanied by improvement in the delirious features, but with a frank manic development. Recovery occurred after hospitalization for six months.

Case 4 is included only because some features of the fully developed psychosis bear a close likeness to those detailed previously. Unfortunately, there is no adequate information available on the prodromal period.

Case 4.—A man, aged 40, married, a railroad conductor, was injured in an accident on Aug. 15, 1927, following which the left leg was amputated. Convalescence apparently was uneventful until August 28, when he had a "sinking spell," which was repeated on August 31. On September 6 he began to be "out of his head all the time," and on the following day he was transferred by ambulance to the Henry Ford Hospital, Detroit.[12]

He was not in contact and talked rapidly with only a little distractibility as a result of auditory stimuli. He moved about in bed, at times shaking the sides vigorously, at times moving his head and hands rhythmically.

12. Dr. Thomas J. Heldt, Chief of the Neuropsychiatric Division, the Henry Ford Hospital, Detroit, furnished the summary of this case.

Physical Examination.—On admission the general physical condition was good: The temperature was 99.8 F. (rectally); the pulse rate, 100; the respirations, 18; the blood pressure, 120 systolic and 80 diastolic. The amputation stump was clean. The red blood cells numbered 3,912,000; the hemoglobin was 15.3 Gm.; the white blood cells numbered 11,160, with 78 per cent polymorphonuclear neutrophils; the blood nonprotein-nitrogen was 32.5 mg., and the sugar, 91 mg. The urine was normal. Examination of the spinal fluid showed: 2 cells; Pandy and the Wassermann tests, negative; colloidal gold and mastic curves, normal.

The following verbatim statements were obtained September 9: "I want some beer—. (Say four three times.) Four, four, four—five—six—seven—eight—nine—ten—eleven—three—seven, three times seven is twenty-one, then we'll drink again. . . . then we'll take another breath—G—d—what am I doing in here? (Notices that we are writing notes) I'll write you a letter—three—five—(throws back the cover and exposes himself)—Floyd—brother—Red—that's myself . . . . three—four—five—that's it, he went back inside. What for? He's the Lord—prayer—two aces, women, women, lovers and women . . . . —(mumbles something faintly) Tom—are you ever going to get through writing? That dam leg ain't bandaged up very good—Tom out in the mud (?). (Makes clucking sound with tongue and lips) Get up—fire engine . . . . I haven't had a drink of whisky for a hell of a long time—what do you have to do to get a drink around here . . . . Sick—operate—Detroit—I want beer. Did you write that down? (Yes) Well, write it three or four times, maybe I can get it—(silent for a minute or two—closes eyes, lies quietly). (Are you sleepy?) Yes. (Do you mind if I ask you some questions before you go to sleep?) No, that's all right. (Are you a married man?) Yes. (Do you have any children?) Three of them. (How old are they?) May took a chance, she was scared—she can make two if she wants to make two—two pencils."

September 10: The patient was talking quite rapidly but did not move about in bed except for an occasional motion with his hands and a slight turn of the head on the pillow. The eyes were held firmly shut. "Who killed that woman? Three times three, there you go. Now try it three times. You roll this time and I'll roll last, Haggerty. Who thought of it first? Seven, I made seven . . . . Back up. Jesus Christ. Who's it for? (Door pushed open.) That G—d—Haggerty come in. Who suffered? Now I forgot something. Bill Luderman. There you are. Red Hahn . . . . Who's crazy now? Nobody. Who was Lutzenheiser? G—d—Jews. Who would be in misery? Nobody. Water. Wine . . . Who would scare a woman? A man. Why didn't you do it yourself? Couldn't. Who wants liver? I swallowed it first. Who cut his leg off? Now who's insane? I had to have a wagon. Who smoked? Mother! it can't be you. Must be a Jew . . . . Who'd be a dog? Rabbits. Three times three is nine. Amie! Martie! Who could be a woman? Oh what he said! Popcorn. What did they need? Three times three. Who lied to me? . . . cattle—that's what we want. Who likes her now? That's a handle. Who wants a crank? Two of them. Nuts. That's easy. Who do I have to work? You damn dumb thing. Don't you have to think? Christ. I will . . . . I was just upside down. Wiggle, wiggle, why don't you wiggle? Who had the fun? Hell it was one. Wiggle. Who had to do it first? Haggerty. . . ."

The patient was in a half-crying state. He moaned, "Who'd take the chances I did? Jesus Christ! Who figured it out? Red Hahn. Well there's five. Who killed that cat? Who's insane? Nuts . . . . (Tone of voice changes to a more common mild quality.) Tommy Warren. Who said seven? Whose name? Now I'll breathe again. What did I do to him? Bumped him hard. Got both

hands cut off. Who wouldn't take a chance? . . . . Had to be two, cuckoo, cuckoo, ain't that crazy? Who said it first. . . . ."

The patient was talking very rapidly, also tossing about in bed, shaking the sides of the crib. ". . . . you lost your job. There's that light. Who lost that airplane? He's a Jew. Who is better looking than Red Hahn? I knowed he liked liquor. I knew he — —. What's two hours in a corn field? . . . . Who wants to grease a wagon twice when he can clean a harness once. Who is the seventh man? Who is the nigger? . . . . I don't like a Jew and never did. I picked all of the cherries in one day. Let's go. Guess it for me. What's elec-tric-i-ty. . . ."

From these statements are noted: (1) push of talk; (2) distractibility (only slight); (3) rhythmic sing-song cadence, with question-answer formula.

Course.—The patient often smiled; at other times he whimpered. He had to be tube fed and offered no resistance. The physical condition was satisfactory, the temperature reaching its peak of 100.2 F. (rectally) on September 10. The pulse rate averaged 90. Sleep was good after the first five days. About September 19 the patient began to improve mentally, and within two days he was oriented for place, time and person, with intact sensorium except for the period of the hospitalization which he said was like a dream and could not be remembered. The mood continued slightly elated.

He was discharged to his home physician on Sept. 22, 1927, showing a pulse rate of 70, and white cells count of 6,320, with 74 per cent polymorphonuclear neutrophils. There was a slight superficial infection of the stump.

Summary.—A middle-aged man in good physical condition suffered an injury which necessitated amputation of the leg. Thirteen days after the operation there were "sinking spells," and nine days thereafter delirium ("amentia"). The latter was characterized by manic-like talk, with a curious rhythmic sing-song cadence and repetitiousness, slight distractibility and restrained rhythmic motor overactivity. The toxic factors were only slightly evidenced in fever, anemia, sleeplessness early in the period of hospitalization and a late superficial infection of the stump.

It was impossible to correlate the topics of the stream of talk with any specific personality or situational factors of importance.

SUMMARY OF CLINICAL CASES

The four cases described are offered with the following points in mind: 1. They justify the term "postoperative psychosis" as defined by Picqué and Kleist. 2. They are undoubtedly dissimilar in many fundamental features (cases 1, 2 and 4 may be classed with the "amentia" of Bonhoeffer, while case 3 appears in the long run to have been manic-depressive). 3. They all show the development of (1) fear of operation or of postoperative treatment; (2) misinterpretations of the attentions of physicians and nurses; (3) growing uncooperativeness because of the threat to the personal safety, and (4) the establishment through this uncooperative attitude of a vicious circle with the factors of infection, fever, dehydration, anemia and cachexia.

In connection with the origins of the factor of fear, the following appear important: 1. Fear of operation is probably present in all. It becomes necessary to try to determine the criteria that should warn one against operation. When fear is marked and cannot be allayed by ordinary reassurance, operation should be delayed, unless an emergency exists.

2. Fear and depression arise from ill-advised expressions used habitually by medical attendants, such as the mention of the nearness of death. Most people do not have the same easy-going attitude toward death as that boasted of by physicians. Casual statements to others of "turns for the worse," and other less pointed references to the condition, which can be overheard and give rise to unwarranted concern, should also be mentioned.

3. The activities of medical assistants who perhaps unkowingly are a source of irritation and of misunderstanding to patients need investigation.

4. The insistence on routine medication when the patient objects to it should make one consider the ultimate usefulness of the procedure.

5. The clear need is seen for taking the patient into one's confidence as much as possible in regard to major (and more minor) procedures. One ought not to give hypodermics, transfusions and infusions without formulating to the patient what is being done in terms that he can accept with confidence.

6. The control of the environment, especially of visitors, is absolutely essential, to keep from the patient disturbing worrisome news and to stress a quiet confidence in the working out of things.

These suggestions are not blanket rules, of course, and must be utilized only with a good idea of the sort of patient one deals with. It should be stressed that personality cannot be taken in at a glance, as some suppose, but a careful survey of the past for evidences of emotional stability or instability (especially fear and depression), idiosyncrasies and twists in thinking and a consideration of the degree of intelligence will furnish a fairly reliable barometer as to the probable activity of the patient at operation.

The cases reported furnish examples of the difficulties occasionally encountered in the surgical practice of doing what seems useful, even absolutely necessary, for the patient in such a way as to secure his whole-hearted cooperation. They show clearly that the best of surgical care can go amiss and lead to complications which pursue a time-consuming course and which can actually endanger the patient's life. They point to the need for a careful self-appraisal of the activities of all concerned in the surgical care of patients, to the end that confidence

may develop in them from mutual cooperation and understanding of all the factors at work in the event.

THE EXPRESSION OF FEAR AND INSECURITY

In these cases fear plays a rôle somewhat different from its usual one. Instead of fear developing into blind panic, there is an accompanying lowered mood (depression) and then elation. Even in the elation, fear is still evident. For example, in case 2, the patient went into an elated, overactive phase when he found himself alive in a hospital instead of about to be buried. Yet at the same time he was suspicious and fearful, crawling under the bed to escape the attendants. I have questioned "normal" people who have undergone anesthesia for an operation, and one woman, an anesthetist, gave an instructive story. She had submitted to her first general anesthetic preliminary to a minor operation for an infected finger. As she succumbed to the anesthetic, she remembers distinctly fearing that the operator would make an incision before she was completely anesthetized. To avert this she kept the finger constantly moving. This incident offers a striking confirmation for the events in case 2, in which the overactivity was designed to prove that the patient was still alive (and one may believe that the same situation existed in case 1).

Much attention has been given to the disorganizing nature of fear (panic) in psychoses. Less has been said of the facts evident in these cases, namely, the significance of fear in bringing into the open strong affective reactions (case 3) which become fixed in a typical manic excitement, after a short period of wavering between initial depression and elation.

The following case of manic-depressive reaction illustrates somewhat similar dynamic factors:

> A man, aged 19, entered a university from a protected rigidly ethical environment, and to his dismay was confronted with sexual and alcoholic excesses (at least with tales of them) on the part of his classmates, which made him feel inferior and insecure. There developed a typical depression, and because of his mental difficulties he had to leave the school at the Christmas holiday. On his return home, he was slow, showed no initiative and was depressed in spirits, but he gradually improved, and with the opening of the second semester (about Easter), he reentered school. He returned with the avowed determination of showing every one that he could be a good sport and still retain his ideals. He promptly went into a full-fledged manic excitement for which he was hospitalized at the Henry Phipps Psychiatric Clinic for several months. He made a complete recovery.

This case is typical of many manic-depressive reactions. The outstanding psychogenic factor is the insecurity of the situation; the onset is with depression, the subsequent swing being to excitement. The time factor offers the principal difference between this case and case 3.

In this case the insecurity and depressive difficulties were cumulative over some months. In case 3, this phase was condensed to a few days, no doubt through the emergency character of the situation. It is as though under the impetus of a serious threat to the personal safety, a serious depressive mood is abbreviated to a very short time.

The elated activity in cases 1 and 2 are also to be linked with the compensatory swings seen after panics. A common experience is the feeling of well-being experienced after painless extraction of a tooth when the event has been anticipated with dread for some time. The persistence of this phase for several days or weeks may result partly from the degree of fear and partly from the toxic state.

STREAM OF TALK

The stream of talk, with its rhythmic, sing-song repetitiousness, and to some degree ease of association, its origins at times from environmental stimuli, yet its relatively weak distractibility, is an outstanding feature.

The origins of the content in environmental stimuli may be in part accounted for by the well known special-sense hyperacuity existing under conditions of fear, in part by the distractibility of elation. Yet the sticking to topics, with little reaction to environmental interjections, is best explained by the relatively greater concern for endogenous content. The outstanding feature is the rhythm, with a question-answer organization, suggesting well known nursery rhymes, or the rhythmic cadences of some of Gertrude Stein's [13] productions. In this connection I again interrogated "normal" people for their reaction under anesthesia, and several remarked that with the induction of anesthesia and on "coming out" thoughts came with a rush, in a rhythmic fashion, "Just as in nursery rhymes." (One subject is responsible for the nursery rhyme comparison given on page 688). One might suspect that this rhythm has a basic grounding in such body rhythms as the pulse (with its loud subjective reverberations resulting from some anesthetics) or respiration. This point cannot be settled with the data at hand.

SUMMARY

1. To date most of the literature on postoperative psychoses has stressed the factors of (1) infection, and (2) constitutional predisposition of the patient.

2. The psychogenic factors have been neglected.

3. Four cases are presented showing: (*a*) late development after operation; (*b*) undoubted mild toxic factors; (*c*) early fear, mistrust

13. Stein, Gertrude: Tender Buttons. Objects, Foods, Rooms, New York, Marie Claire, 1914.

and depression resulting from the course of the postoperative events, and (*d*) later swing to elation and overactivity, in which fear was still much in evidence.

4. In three cases the elation was rather restrained, with a stream of talk showing push, a sing-song rhythmic delivery, repetitiousness, little distractibility, some flight and recurrent topical content. The other case showed typical manic flight and overproduction.

5. The relation of fear in this setting to more typical affective reactions and to disorganizing panic is discussed. The time factor seems to be the important differential point separating these reactions from the more typical manic-depressive psychoses. These cases showed no features of disorganization.

6. A plea is made for close self-scrutiny in all contacts with patients after operation in order that the plastic factors of the situation may be utilized for the patients' interest, through the development of mutual cooperation and confidence.

HEAT PRODUCTION AND HEAT CONTROL IN THE SCHIZOPHRENIC REACTION

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Of the numerous attempts to reveal physiologic change associated with reaction in the schizophrenic group, one of the most consistently successful has been that concerned with basal metabolism or, as Hoskins [1] more correctly designated it, heat production. According to numerous workers, heat production among schizophrenic patients is reduced, Hoskins' average for 214 male patients with schizophrenia being 88.3 per cent of normal. Walker [2] found that 50 per cent of thirty patients with schizophrenia had an average basal metabolic rate of —20, the others giving values within normal limits or slightly above it. Numerous other workers have reported similar findings (Bowman and Fry [3] and Whitethorn and Tillotson [4]). The chief difficulty has been in securing sufficient cooperation to obtain the relaxation necessary for accurate results, so that the figures should probably be lower than those stated.

On accepting the previous work on heat production as being reasonably well authenticated, the question arises, first, as to the effect the diminished heat production in schizophrenic patients has on their body temperature, and, second, as to whether the mechanisms of heat control are called on to compensate for the lowered output of heat. At this point it will be of value to review the mechanisms of heat control and heat production.

On exposing the organism to extremes of cold, both groups of mechanism are brought into action to preserve the normal heat balance of the body. Heat production is increased by increased muscular tension and, if this fails, by dint of increasing oxidation more drastically by throwing large masses of muscles into action by shivering. It is probable that the epinephrine content of the blood is also increased. The

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From the Brandon Hospital for Mental Disease.

1. Hoskins, R. G.: Oxygen Consumption (Basal Metabolic Rate) in Schizophrenia, Arch. Neurol. & Psychiat. **28**:1346 (Dec.) 1932.

2. Walker, J.: Basal Metabolism in Mental Disorders, Especially Dementia Praecox, and the Influence of Diathermic Current on the Same, J. Ment. Sc. **70**: 47, 1924.

3. Bowman, K. M., and Fry, C. C.: Basal Metabolism in Mental Disease, Arch. Neurol. & Psychiat. **14**:819 (Dec.) 1925.

4. Whitethorn, J. C., and Tillotson, K. J.: Oxygen Consumption in Dementia Praecox, Boston M. & S. J. **192**:1254, 1925.

mechanisms of heat control are also brought into action. These are largely, if not entirely, dependent on the sympathetic nervous system, and involve, on exposure to cold, the constriction of the cutaneous vessels. By this means the skin is made to perform its action as insulator. When the internal temperature of the body is in danger of rising, the processes are largely reversed, although it is to be noted that heat production does not respond nearly so sharply to increased heat as to increased cold; indeed, Martin [5] found that exposure to tropical temperatures was required for several days before his basal metabolic rate showed a decided drop. The body is more concerned with protection against the loss of heat than with the effects of excessive heat. In recent years Barbour [6] described a mechanism for the further control of heat consisting in the transudation of fluid from the cutaneous vessels in response to increased cold, the transudation producing a body of fluid in the tissue spaces of the skin which acts as an insulator. Also, in consequence of the decreased volume of blood, less heat is brought from the deeper tissues into the circulating blood. When the internal temperature is high the process is reversed. The fluid from the cutaneous tissue spaces reenters the blood stream, the volume of blood is increased again, and in consequence more heat is brought up from the deeper structures.

EXPERIMENTAL WORK

Coming now to an actual consideration of the problem of heat control and schizophrenia, I shall place a number of questions which I have striven to render as simple and unequivocal as possible.

QUESTION 1.—*Is there any evidence that the internal temperature of schizophrenic persons living at room temperature (from 68 to 72 F.) differs from that of other types of patients with mental disease under similar conditions?*

The oral and axillary temperatures, recorded with certified thermometers, were taken as giving an index of the internal and cutaneous temperatures, respectively. The temperatures of fifty schizophrenic and fifty nonschizophrenic patients, the latter comprising representatives from all psychotic groups with the exception of the schizophrenic—senile, arteriosclerotic, and drug addicts—were recorded every hour between 7 a. m. and 6 p. m. for three days. The average oral temperature of the groups for each hour was then charted. During this period the patients lived at room temperature, and the amount of exercise was cut to a minimum. The results are seen in chart 1. Each reading represents the average of 150 records of temperature. It can be seen that the average internal temperature of the schizophrenic group is slightly lower than that of the nonschizophrenic group.

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5. Martin, C.: Thermal Adjustment of Man to External Conditions, Lancet **2**:561 (Sept. 13) 1930.

6. Barbour, H. G.: Heat Regulation and Water Exchange: VII. Evidence That Cold Anhydremia Is Due to Loss of Fluid from the Blood Stream, Am. J. Physiol. **73**:320, 1925.

QUESTION 2.—*What evidence is there that the mechanism of heat control, as distinct from the mechanism of heat production, acts differently in schizophrenic persons in the production of these temperatures?*

It is clear that in the production of any given body temperature two factors are operative—heat production and heat control. If the heat production is low,

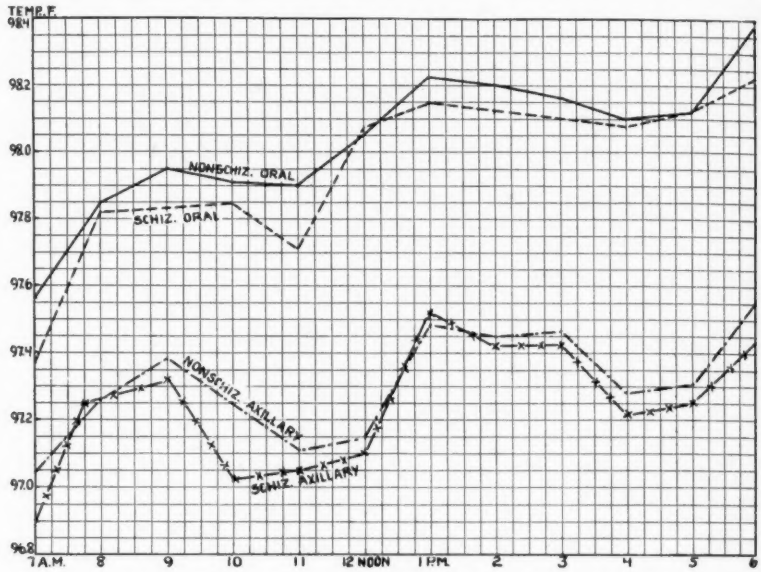

Chart 1.—A record of the average oral and axillary temperature for groups of fifty schizophrenic and fifty nonschizophrenic patients. The records were taken over three days; the curves are based on 7,200 readings.

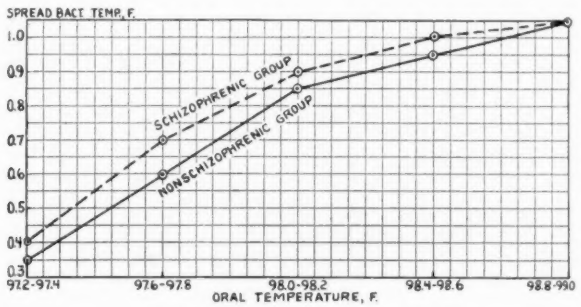

Chart 2.—The temperature interval from 97.2 to 99 F. The curves are based on 1,180 readings.

it may be that heat control will exercise a compensatory function to preserve the body temperature at a nearly normal level. It is also clear that if the mechanisms of heat control are functioning actively to compensate for inadequate heat production, the skin will be functioning as an insulator, less heat will be passing through, and therefore the cutaneous temperature will be lower in relation to the oral temperature than usual.

The axillary temperatures of the two aforementioned groups were therefore investigated in a manner similar to the internal temperature. Attempts to demonstrate a difference between the schizophrenic and nonschizophrenic internal (cutaneous) temperature intervals by reference to the curve (chart 1) were largely frustrated by the fact that the temperature of schizophrenic persons apparently responds differently to the ingestion of food than does that of nonschizophrenic persons. Accordingly, a range of oral temperatures from 97.2 to 99 F. was decided on, and the average cutaneous temperature corresponding to each 0.2 degree F. of internal temperature was plotted by a method of sampling whereby each of the fifty patients in each group contributed at least one and not more than three readings to each point in the curves for cutaneous temperature. Any particular cutaneous temperature represented the average of 118 recordings. Chart 2 shows the difference between the internal and cutaneous temperatures in each group. As will be seen, the interval is clearly larger in the case of the schizophrenic group, indicating that the mechanism of heat control is more active in the schizophrenic group than in the nonschizophrenic group.

In considering this group of data the following points must be borne in mind: First, as already mentioned, any given internal temperature is the result of heat production and heat control. From studies in basal metabolism it is clear that the amount of heat produced varies from the basal rate by far the most frequently in an upward direction. Reduction in the basal rate is associated for the most part with grave bodily disturbances, as with myxedema and pituitary dysfunction. The normal heat production of the body in the presence of adequate heat control is definitely more than sufficient to preserve the body temperature within normal ranges. The body even at rest produces 1.2 large calories per minute (Martin). In these markedly subnormal temperatures, as distinct from the slight relative subnormality of the temperatures of schizophrenic persons with their well marked internal (cutaneous) temperature interval, I thought that I was dealing with a partial failure of heat control. That this is true is borne out by the fact that as the temperatures in chart 2 rise toward normal ranges, the internal (cutaneous) temperature interval becomes larger; i. e., the mechanism of heat control is becoming more active and the skin is functioning more completely as an insulator.

QUESTION 3.—*How do the mechanisms of heat control in schizophrenic persons act on exposure to extremes of temperature?*

Cannon has shown that even when the mechanisms of heat control are profoundly disturbed by the destruction of the sympathetic nervous system and even of the suprarenal bodies, relatively little change in temperature is to be noted in the monkeys thus operated on so long as they are kept at room temperature. As soon as they are exposed to extremes of temperature, however, they show serious

aberrations of temperature. To answer the question, twenty-five schizophrenic and twenty-five nonschizophrenic male patients were placed in a tub at 28 C. (82.4 F.) on three different days. The oral temperatures prior to immersion and at three minute intervals for twelve minutes during immersion were recorded. The results of the three tests were combined to form a curve. At this point it should be emphasized that to obtain consistent results the greatest care is necessary. The patients must have been recumbent for at least an hour before immersion. Immersion should be carried out at least two hours after a meal, and the oral temperature should be steady to 0.1 degree F. for nine minutes before immersion. Contrary to expectation, the most usual reaction to immersion in water at 28 C. is a rise of temperature, this apparently being in the nature of an excessive compensatory reaction to the stimulation of the skin.

Four types of reaction were recognized: type 1, in which the temperature subsequent to immersion was throughout higher than the temperature before immersion; type 2, in which there was an initial fall followed by a subsequent rise;

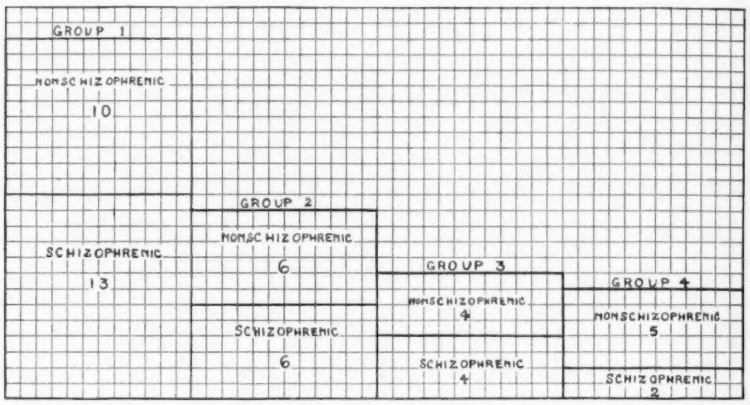

Chart 3.—The distribution of types of reaction to cold among schizophrenic and nonschizophrenic groups of twenty-five each. The curves are based on 750 readings.

type 3, in which the temperature after immersion was throughout lower than before immersion, and type 4, in which there was included a small miscellaneous group. The distribution of the types in the two groups is shown in chart 3. The average reaction of temperature of the two groups was:

	3 Min.	6 Min.	9 Min.	12 Min.
Schizophrenic	+0.02	+0.16	+0.24	+0.20
Nonschizophrenic	—0.04	+0.17	+0.18	+0.16

As will be seen, the reaction of the schizophrenic group to the stimulus of extreme cold is better than that of the nonschizophrenic group. As it is known that the heat production in this type of psychotic reaction is diminished (chart 1) it must be concluded that the mechanism of heat control is more efficient in this group. It seems reasonable that in an organism defective in the capacity for heat production the center for heat control might be more sensitive and react more strongly to cold stimuli in an effort to protect itself from the consequences of the inadequacy.

In testing the reaction to extreme heat, twenty-five of each group were exposed to temperatures ranging from 36 to 42 C. (96.8 to 107.6 F.) for one hour. The patients were in a passive state before exposure. The oral temperature was recorded for four minutes. To overcome the influence of varying degrees of humidity the patients were exposed in small groups composed of equal numbers of schizophrenic and nonschizophrenic patients. The response of the members

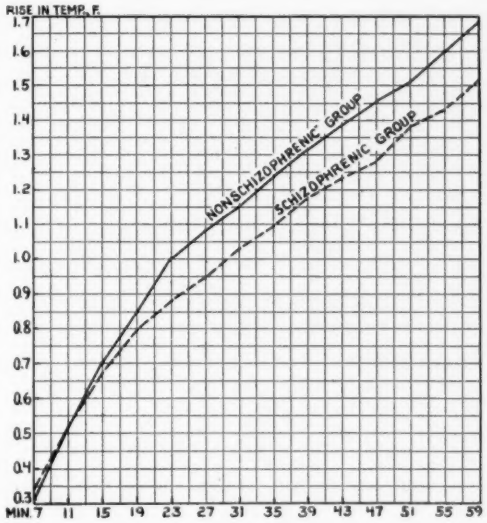

Chart 4.—The average rise of temperature in schizophrenic and nonschizophrenic groups of twenty-five on exposure to temperatures from 36 to 42 C. The curves are based on 750 readings.

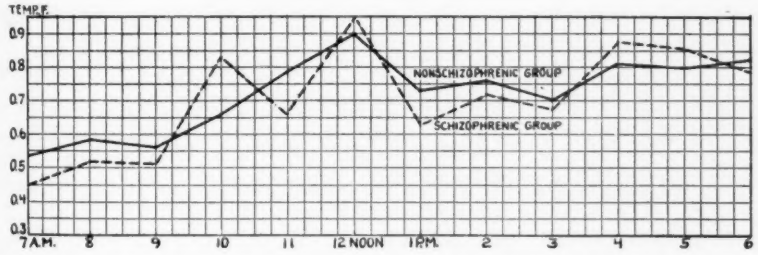

Chart 5.—The oral-axillary interval of schizophrenic and nonschizophrenic groups from 7 a. m. to 6 p. m. The curves are based on 7,200 readings.

of the small groups were, of course, comparable with each other, as were the combined responses of the large groups of twenty-five. The results were much as was expected (chart 4). In consequence of the greater heat production in the nonschizophrenic group the rise in temperature both in comparing the members of the small group with each other and in comparing the combined groups were definitely greater.

It can be deduced from these data that the mechanisms of heat control are not inadequate in schizophrenic patients since they are capable of maintaining the advantage which the lessened heat production affords them. In chart 4 it will be observed that both groups absorb heat for the first fifteen minutes very much as do inanimate objects; then as the heat control begins to function the disadvantage of the increased heat production in the nonschizophrenic groups becomes increasingly apparent. This incidentally offers a corroboration of Martin's findings in regard to the slowness with which heat production adjusts itself to increased external heat as compared with the rapid response to increased external cold. Finally, during investigation it became apparent that the heat response to the ingestion of food was different in schizophrenic from that in nonschizophrenic patients. Heat production occurs more rapidly after a meal, but is not so well maintained (chart 1). Meals are served at 7 a. m., 11 : 30 a. m. and 4 : 45 p. m. No explanation of these two phenomena can be afforded at present. As an index of the response of the mechanisms of heat control to this irregular heat production, the wide fluctuations of the oral-axillary spread in the schizophrenic group as compared with the nonschizophrenic group is notable (chart 5).

COMMENT

The results obtained by observing the temperatures of schizophrenic and nonschizophrenic groups afford confirmation of the statement that heat production in the schizophrenic reaction is low. It was further shown that the mechanisms of heat control are slightly more active in a schizophrenic group than in a nonschizophrenic group. This overactivity was sufficient to compensate for the poor heat production even on exposure to extreme cold. One gains the impression of an intact and active mechanism of heat control in the schizophrenic reaction. This is of special interest because mechanisms of heat control depend largely on the sympathetic nervous system, and one still observes in the literature attempts to correlate the schizophrenic reaction with vagotonia.

CONCLUSIONS

The following observations are based on approximately 10,000 temperature readings in fifty schizophrenic and fifty control subjects:

1. The internal temperature in the schizophrenic group was throughout the day slightly lower than that in the nonschizophrenic psychotic group, both groups being at room temperature.

2. There is evidence to show that at room temperature the mechanism of heat control in the schizophrenic group is more active than in the nonschizophrenic group.

3. Findings on exposure to extreme cold suggest that the mechanism of heat control acts more sharply and more strongly in the schizophrenic than in the nonschizophrenic group.

4. Exposure to extreme heat reveals no weakness in the mechanism of heat control in the schizophrenic group.

5. The response to the ingestion of food is more marked but less well sustained in the schizophrenic than in the nonschizophrenic group.

6. These findings are not consistently true for the individual subject, but only for the group. It is doubtful whether it would be wise to accept the suggestion afforded by any one of the experiments alone, but the fact that all tend to reveal the same condition, together with the large number of observations, make one feel justified in according a fair degree of probability to the contentions that (*a*) heat production in the schizophrenic is lower than in the nonschizophrenic group, and (*b*) heat control in the schizophrenic group is more active than in the nonschizophrenic group, this being apparently in the nature of a compensation for the decreased output of heat.

CARDIOVASCULAR SYSTEM IN SCHIZOPHRENIA STUDIED BY THE SCHNEIDER METHOD

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The Schneider test, which purports to afford a measure of cardiovascular "fitness," was elaborated [1] from a somewhat similar test devised by McCurdy. Its purpose was to aid the flight surgeon in judging the degree of physical fitness of military aviators, particularly in regard to such functional aspects as fatigue, staleness and vasomotor instability. It is reported to have given satisfaction and to be the best test devised in aviation medicine for the measurement of physical fitness and vasomotor stability.[2]

TECHNIC

The test is carried out as follows: The subject first reclines for five minutes.

1. The heart rate is then counted for twenty seconds. When two consecutive twenty second counts are the same, the count is multiplied by 3 and recorded.

2. The systolic pressure is taken two or three times by auscultation and recorded.

The subject then rises and stands for two minutes, to allow the pulse to assume a uniform rate.

3. The heart rate is counted until two consecutive fifteen second counts are the same, when it is multiplied by 4 and recorded. This is the normal standing rate.

4. The pulse rate taken while standing minus that while reclining gives the "increase on standing."

5. The systolic pressure is taken as before and recorded.

6. Timed by a stop-watch, the subject steps on a chair 18 inches (45.72 cm.) high five times in fifteen seconds. To make this uniform he places one foot on

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From the research service of the Worcester State Hospital and the Memorial Foundation for Neuro-Endocrine Research.

1. (*a*) Schneider, E. C.: A Cardiovascular Rating as a Measure of Physical Fatigue and Efficiency, J. A. M. A. **74**:1507 (May 29) 1920. (*b*) Schneider, E. C., and Truesdell, D.: A Statistical Study of the Pulse Rate and Arterial Blood Pressure in Recumbency, Standing, and After a Standard Exercise, Am. J. Physiol. **61**:429, 1922.

2. (*a*) Bauer, L. H.: Aviation Medicine, Baltimore, Williams & Wilkins Company, 1926. (*b*) Scott, V. T.: The Application of Certain Physical Efficiency Tests, J. A. M. A. **76**:705 (March 12) 1921.

the chair at the count "one." This foot remains on the chair and is not brought to the floor again until after the count "five." At each count he steps onto the chair, and at the word "down" replaces one foot on the floor. This should be timed accurately so that at the fifteen second mark on the stop-watch both feet are on the floor.

7. The pulse rate is counted immediately at the fifteen second mark on the stop-watch and counted for fifteen seconds. The total is multiplied by 4 and recorded.

8. The pulse rate is counted by fifteen second periods until it has returned to the normal standing rate. The number of seconds it takes for this to return to normal is recorded. In computing this return, the count is from the end of the fifteen seconds of exercise to the beginning of the first fifteen second count in which the pulse rate is normal. If the pulse rate has not returned to normal at the end of two minutes, the number of beats above normal is recorded and the counting discontinued.

9. The points are then checked and the final rating entered.

10. Special note is made of the amount of sleep, amount of smoking, kind of work (outdoor or indoor, active or sedentary), time since the last meal, any personal worries or any pathologic condition which might affect the conditions of the subjects.

This technic is adapted from Schneider.[1a]

The results of the foregoing procedures are scored as indicated in table 1, the final rating being determined by adding the subscores.

Schneider [1a] emphasized that to insure reliable results the subject must be mentally at ease during the test, that carelessness in determining the pulse rate immediately after exercise is especially to be deprecated and that exactly the same procedure must be followed at all times. He emphasized the need of observing precautions during a two hour period before the test. During this time no food is to be taken, no tobacco used and no vigorous exercise permitted. He also pointed out that temporary indispositions (diarrhea, toothache, flatulence and indigestion), recent or impending illness, loss of sleep and warm baths materially lower the score. Discontinuance of training and all exercise after a season on the football squad was found to result in a lowering of the score, while regular, systematic, healthful exercise tended to improve it, although it might be as long as a month before definite improvement occurred, and in persons who used alcohol or kept late hours the improvement was not as marked as that ordinarily expected.

The highest score possible is plus 18, and the lowest, minus 11. Bauer [2a] graded results roughly as follows: from 14 to 18, excellent; from 11 to 13, very good; from 9 to 10, fair; from 7 to 8, doubtful and below 7, unsatisfactory. Both Bauer and Scott [2b] noted that bradycardia gives a better Schneider index rating than the condition warrants, but did not specify to what extent.

PREVIOUS RESULTS

The findings previously reported on normal subjects demand consideration for the criteria they afford for the interpretation of results in psychotic patients. Schneider [1a] reported a rather high correlation between the test scores and the results of independent clinical examinations. It appeared that when the score was 9 or less the clinician commonly found something wrong. He [1b] also reported a study of diurnal changes in the index of 7 normal men and 7 normal women at sedentary occupations. The mean score for the men for twenty-four hours ranged from 10.1 to 13.6, and for the women from 9 to 14. The largest depressions

TABLE 1.—*Points for Grading Cardiovascular Changes*

A. Pulse Rate When Reclining		B. Increase in Pulse Rate on Standing				
Rate	Points	0-10 Beats, Points	11-18 Beats, Points	19-26 Beats, Points	27-34 Beats, Points	35-42 Beats, Points
50-60	3	3	3	2	1	0
61-70	3	3	2	1	0	−1
71-80	2	3	2	0	−1	−2
81-90	1	2	1	−1	−2	−3
91-100	0	1	0	−2	−3	−3
101-110	−1	0	−1	−3	−3	−3

C. Pulse Rate on Standing		D. Increase in Pulse Rate Immediately After Exercise				
Rate	Points	0-10 Beats, Points	11-20 Beats, Points	21-30 Beats, Points	31-40 Beats, Points	41-50 Beats, Points
60-70	3	3	3	2	1	0
71-80	3	3	2	1	0	0
81-90	2	3	2	1	0	−1
91-100	1	2	1	0	−1	−2
101-110	1	1	0	−1	−2	−3
111-120	0	1	−1	−2	−3	−3
121-130	0	0	−2	−3	−3	−3
131-140	−1	0	−3	−3	−3	−3

F. Return of Pulse Rate to Standing Normal after Exercise	
Seconds	Points
0- 30	3
31- 60	2
61- 90	1
91-120	0
After 120: 2 to 10 beats above normal	−1
After 120: 11 to 30 beats above normal	−2

F. Systolic Pressure, Standing, Compared with Reclining	
Change in Mm.	Points
Rise of 8 or more	3
Rise of from 2 to 7	2
No rise	1
Fall of from 2 to 5	0
Fall of 6 or more	−1

in the score seemed to follow the taking of meals. The lowest level for the day was at 2:00 p. m. and the highest at 2:00 a. m.

Scott [2b] applied the test to 410 candidates for aviation. The mean score was 11.078; the maximum, made by 4 men, was 18, and the minimum, made by one man, —3. A score below +10 was recorded for 34.95 per cent of the candidates. It appeared that with a Schneider index of 7 or less, the neuropsychiatrists would find at least three of the following symptoms: (1) increased psychomotor tension; (2) prolonged secondary dilatation of the pupils; (3) relaxed peripheral circulation, or (4) tics and tremors. A score of 7 or less, he stated, indicates improper functioning of the neurocirculatory apparatus and a physical condition rendering flying unsafe, while a score of 8 or less establishes an indication for a physical examination.

Reed [3] reported as the "average" score for 30 male employees of a state hospital +10.6, with 43.4 per cent scoring below 10; for 36 female employees, +9.05, with 61.2 per cent scoring below 10.

Griffith and his associates [4] attempted to evaluate seasonal fluctuations; their data were rather inconclusive, but suggested that the score is highest in the summer. In women it appeared to fluctuate slightly with the menses.

The indication of the foregoing evidence is that the test is a fairly sensitive indicator of "fitness," but that its interpretation demands a careful consideration of attendant circumstances.

Trentzsch [5] seems to have been the first to report on the application of the Schneider test to psychotic patients. His data, based on observations on 124 white males, show a mean score for the entire schizophrenic group (85 patients) of 8.4 with 57.6 per cent scoring below 10. For the subgroups, the mean scores were: paranoid, 10.5; mixed, 11.3; catatonic, 6, and hebephrenic, 6.3. For 9 manic-depressive patients the mean score was 12.5; 11.1 per cent scored below 10. Trentzsch's data are consistent with those reported by Lewis [6] from the same hospital (St. Elizabeth's) on the weight of the heart. Paranoid and manic-depressive patients tended to have normal or above normal heart weights; catatonic and hebephrenic patients, subnormal heart weights. By roentgen study Trentzsch noted also a higher percentage of "tubular" hearts in the catatonic and hebephrenic than in the paranoid patients. He commented with what seems considerable reason, on the relationship of these findings to the types of psychotic reaction: The catatonic and hebephrenic groups represent "purely regressive," and the paranoid and manic-depressive "compensatory projecting reactions." Trentzsch noted that the paranoid patients showed some improvement of score with a month's training by standard exercises, but the catatonic and hebephrenic patients showed no response. The number of patients (25) and the duration of the training given, however, seem too inadequate to permit definite conclusions.

Reed [3] reported the results of Schneider tests on 153 male and 122 female schizophrenic patients. His results for the males as a group were similar to those of Trentzsch, but by subtypes they were markedly different. The mean score for all the male patients was 9.5, with 56.1

3. Reed, R. G.: Circulatory Rating and Pulse Pressure in Schizophrenia, State Hosp. Quart. **12**:34, 1926.

4. Griffith, F. R., Jr.; Pucher, G. W.; Brownell, K. A.; Klein, J. D., and Carmer, M. E.: Studies in Human Physiology: II. Pulse Rate and Blood Pressure, Am. J. Physiol. **88**:295, 1929.

5. Trentzsch, P. J.: The Objective Findings in the Psychoses, Arch. Neurol. & Psychiat. **12**:370 (Oct.) 1924; personal communication to the authors, 1933.

6. Lewis, N. D. C.: The Constitutional Factors in Dementia Praecox, Monograph 35, Washington, D. C., Nervous and Mental Disease Publishing Company, 1923.

per cent scoring below 10. His "normal" average, it will be recalled, was only slightly higher, viz., 10.6. By subtype the mean scores were: paranoid (49 patients), 9.2; catatonic (21 patients), 11.3, and hebephrenic (85 patients), 9.4. Thus, only the catatonic patients gave a score significantly different from the average, and this was elevated.

Reed also made what seems a valid subdivision into indolent and active groups. The indolent patients (54) gave a mean score of 8.5, 68.6 per cent scoring below 10, and the active patients (99) scored 10.1, 48.6 per cent giving scores below 10.

None of the investigators except Griffith and his associates [4] seem to have observed the precautions outlined by Schneider for obtaining valid scores. Scott's work was done before these precautions were published. Trentzsch stated that he did not observe the two hour control period prior to the test. Reed's tests were made from one to three hours after dinner. Failure to observe the two hour interval would presumably have tended to give scores lower than those considered valid by Schneider.

PERSONAL OBSERVATIONS

Except as will be indicated, Schneider's technic was rigorously followed in our tests. Twenty-five male staff members acted as controls. This group ranged in age from 22 to 40 years, but mostly between 22 and 30 years. All seemed to be in good general health except for passing minor indispositions in 3. Calculation showed that eliminating these 3 would have given only a slightly and insignificantly higher average score. The fact that they were habituated to an environment similar to that of the patients was an obvious advantage.

The group of patients included 99 persons suffering from schizophrenia, from whom 311 fairly satisfactory scores were obtained.

Since representativeness of the scores and their significance in comparison with those of other investigators depend essentially on concomitant circumstances, it seems incumbent to record these in detail.

All the cases were unanimously classified both by the hospital and by the research staffs as presenting the schizophrenic reaction. Subtyping offered a difficult problem, as it does in any well studied schizophrenic population. Cases classified as paranoid seemed fairly typical, although some represented a postcatatonic paranoid condition; usually the psychosis was in a rather quiescent state. The catatonic patients seemed fairly typical, but at the time of the tests most of them had to some extent recovered. Those classified as hebephrenic often seemed atypical; schizophrenia unclassified, with hypomanic coloring, would have fairly characterized some of them. The mixed group showed in various degrees and combinations coincident manifestations—catatonic, hebephrenic, etc. The group included under "unclassified" seemed to have a disorder not fitting strictly into any of the classic subgroups, but definitely schizophrenic. The indeterminate group was made up of patients in whom the disorder had lasted a long time, and who for some time before the tests presented little of the distinctive coloring of a subtype.

The general clinical state of the patients at the time of the test was recorded in terms of the scheme of Erickson and Hoskins.[7] Patients in grades A, B, C and D contributed 72 per cent of the total tests. These, by definition, represented patients in relatively good condition. They were up and about, working fairly well. A small proportion were in a sufficiently disturbed condition to be receiving hydrotherapy daily, or were sufficiently stuporous to need feeding by a tube.

The mean age of the patients was 31.6 years, with a maximum of 52 years and a minimum of 15 years. The average duration of the psychosis was 7.9 years, the minimum 4 months and the maximum 24 years. The mean time since the first admission was 5.9 years, the maximum 24 years and the minimum 1 month. The mean weight of the group was 136 pounds (61.7 Kg.); by the Metropolitan Life Insurance Company height-weight-age standards they averaged 10 per cent below prediction. The maximum nutritional index was 141.2 per cent and the minimum 71.8 per cent. Roughly, about 75 per cent were underweight and 25 per cent were of ideal weight or above, according to these standards.

Four per cent showed visceroptotic posture; 5 per cent, varicose veins; 2 per cent, hemorrhoids; 2 per cent, varicocele; 53 per cent, cyanosis of the extremities, with or without coldness or clamminess; 12 per cent, cardiac murmurs (probably all functional); 4 per cent, hypostatic pitting edema and 1 per cent, nonpitting edema of the ankles; 5 per cent, systolic blood pressure over 140 mm.; 5 per cent, systolic bruits, thrills or unusual pulsations over the arteries; 1 per cent, the heart small to percussion, and 1 per cent, the heart enlarged to percussion.

Prior to hospitalization 5.5 per cent of the patients had been engaged at sedentary work, 54.6 per cent at fairly active work and 39.8 per cent at manual labor. At the time of the tests practically all the patients were up and about from eleven to fifteen and a half hours daily; they walked at least 1,400 meters, and climbed up and down at least 8 meters of stairs a day. In addition, from 70 to 80 per cent were engaged in daily industrial occupation for from three to seven hours a day, most of it manual labor on the grounds, in the kitchen, in the cafeteria or elsewhere. Even the 20 to 30 per cent left in the wards, except in a few instances, pushed from 5 to 7 Kg. of polishers up and down the floor for from one to four hours daily.

Investigation of the patients' histories brought out that only 8 per cent had used alcoholic beverages to the extent of over two glasses of whisky or five glasses of beer a week; 66 per cent had been nearly totally abstinent prior to hospitalization. In the hospital abstinence was required. According to the nurses' records, the patients used an average of about four or five cigarets a day. The mean period of sleep for a month prior to the test as recorded by the nurses was about eight hours a night, and for the night before the tests, from 7.5 to 7.9 hours.

In certain respects minor deviations from Schneider's prescribed routine crept in. In 33 tests the pulse rate was followed after exercise only until it had dropped to or below the standing rate. It was then observed that sometimes the pulse showed a secondary increase after having reached the standing rate. Thereafter it was determined throughout two minutes as a routine, and if a second increase occurred allowance was made, a lower score being given. About nine tenths of the tests were made between 2:30 and 5:00 p. m., and one tenth between 10:00 a. m. and noon.

In most cases the tests were carried out with only one observer in the room, but in about 55 two observers worked together, one taking blood pressure and

7. Erickson, M. H., and Hoskins, R. G.: Grading of Patients in Mental Hospitals as a Therapeutic Measure, Am. J. Psychiat. **11**:103, 1931.

the other observations on the pulse rate. Usually there were three or four, sometimes six or eight patients in the room awaiting their turns. The room in which the tests were made was exposed to disturbances from the adjacent ward. Patients occasionally disturbed each other when a group to be tested was in the same room. Each patient walked from fifteen to twenty steps just before lying down for the rest period preceding the reclining observations. Less responsive patients, some with the catatonic type of psychosis, sometimes had to be stimulated by pulling on their arms to make them step up properly in the exercises. The time of the exercise part of the test was often not exactly fifteen seconds, owing to difficulty in inducing the patients to cooperate precisely, the exercise often being completed in twelve or thirteen seconds, rarely ten seconds, instead of fifteen. The count of the pulse rate, after exercise stopped, was started precisely fifteen seconds after the exercise started, rather than when the exercise stopped. This occasionally brought in a delay of two or three, more rarely four or five seconds in starting the count after exercise.

Except rarely, patients went through the tests without a show of emotional disturbance. In a series of 520 attempts at executing the test, 4.2 per cent had to be discontinued because of the lack of cooperation.

No tests were included in the analysis in which there intervened: (1) very hot weather; (2) detected emotional disturbances in connection with the test itself; (3) poor cooperation and restlessness during the reclining period of the test; (4) physical illness, and (5) the exercise part of the test taking longer than fifteen seconds. There were a number of discards because of physical illness. No systematic medication was administered during or within two months prior to the tests. The fact that cooperation is necessary to carry out the test resulted in a certain amount of selection; we were unable to utilize patients in important schizophrenic states, such as marked uncooperativeness, stupor and excitement. It is not unlikely that a deceptively unrepresentative degree of normality was thus registered.

The results of the study are set forth in tables 2 and 3. The scores and statistical constants are given in table 2. The means, maxima and minima of the items on which the index is based (pulse rate while reclining, etc.) are given in table 3. Data of the patients and controls are given in the same tables for comparison. The scores were analyzed statistically in a variety of ways. It was found that the mean of the first scores and the mean of mean scores for each patient were nearly the same; both were about midway between the mean of the highest scores and the mean of the lowest scores. No characteristics were brought out by the various analyses that are not sufficiently indicated by the distributions presented in the tables.

The mean score for the control subjects was 13.4. The mean of mean scores of each patient was 11.9; the mean of maximum scores was 14.2 and of minimum scores 9.5; the means by last score, by each score and by different types of mean scores came out essentially the same, tending to approach 12 (11.8 to 12.3). The mean of first scores was a little higher, 12.7, probably owing to the fact that the patients happened to be getting relatively more exercise at the time of those

particular tests than was usually the case. The consistency shown when different criteria of selection were employed strongly validates the findings.

The patients as a group scored significantly, though not greatly, lower than the controls (11.9 and 13.4, respectively). Twenty per cent

TABLE 2.—*Schneider Indexes of Control and Schizophrenic Subjects by Total Groups and by Subgroups*

	Number of Subjects	Number of Observations	Range	Mean* (1)	Mean* (2)	Per Cent Scoring Below 10 (1)	Per Cent Scoring Below 10 (2)
Controls	25	30	8 to 17	13.08	13.37 ± 0.54	20	17
Patients							
Total group	99	311	−2 to +18	12.00	11.93 ± 0.20	16	23
Simple	6	17	2 to 16	11.74	11.47	40	29
Paranoid	17	56	5 to 17	12.61	12.27	13	21
Hebephrenic	19	75	4 to 18	11.81	11.59	11	30
Catatonic	14	33	4 to 17	11.59	12.06	17	15
Mixed	9	29	5 to 18	12.72	12.79	0	10
Unclassified	21	53	−2 to +17	11.77	11.96	22	26
Indeterminate	13	48	2 to 17	12.00	11.60	18	21

* The mean for the control subjects is (1) the mean of the first score of each subject and (2) that of all scores. The mean for the schizophrenic patients is (1) the mean score of each patient with two or more scores, and (2) the mean of all scores.

TABLE 3.—*Items of Schneider Indexes for Controls and Schizophrenic Patients*

	Means*		Maximum†		Minimum‡	
	Controls	Patients	Controls	Patients	Controls	Patients
Pulse rate						
Reclining	66.6	63.3	84	102	48	45
Standing	85.0	81.9	100	116	64	52
Change from reclining to standing	+18.4	+18.6	+33	+55	+5	−4
Pulse rate after exercise						
First 15 seconds	96.5	96.7	116	136	80	64
Second 15 seconds	91.4	89.2	108	128	68	52
Third 15 seconds	85.9	85.9	100	128	64	56
Fourth 15 seconds	84.0	82.3	104	124	64	56
Fifth 15 seconds	81.5	80.2	96	120	60	52
Sixth 15 seconds	81.8	79.7	100	124	60	44
Seventh 15 seconds	82.2	79.4	100	120	64	48
Eighth 15 seconds	81.4	79.4	96	124	64	52
Change from standing to first 15 secs.	+11.5	+14.7	+24	+40	0	−8
Systolic blood pressure§						
Reclining	115.5	114.9	133	186	96	82
Standing	120.9	119.0	140	182	96	90
Change from reclining to standing	+5.5	+3.3	+24	+42	−6	−20

* The means for the controls are the means of the first tests, and for the patients are the means of the means of all the tests of each patient (two or more tests).
† The maxima include the means of all observations.
‡ The minima include the means of all observations.
§ In computing the change in pulse rate and systolic blood pressure from reclining to standing position, the maximum means the greatest rise and the minimum the greatest fall.

of the normal subjects received scores below 10. Depending on the different methods of analysis, the proportion of patients scoring below 10 points varied. It was 6 per cent for maximum scores, 42 per cent for minimum scores and 23 per cent for all scores. Thus it appeared that low scores were distributed in essentially the same proportion among patients and

controls, but whereas the lowest individual score of any normal subject was 8, several patients received a considerably lower rating, — 2 being the minimum.

The results were further analyzed according to subtype, according to the ward grade as defined by Erickson and Hoskins [7] and according to the type of occupation previous to hospitalization. The differences were slight, variable and, except as to ward grades, inconsistent. Apparently, after the patient becomes adjusted to the hospital, little of his previous training carries over.

The general similarity between the scores of the patients and those of the controls is paralleled by the similarities of functions set forth in table 3. The pulse rates of the patients averaged slightly lower while reclining at rest (63.3 : 66.6), and in general this difference was maintained during exercise. The spread between the upper and lower values, however, was greater in the patients, as is true of metabolic and physiologic functions generally.[8] The systolic blood pressure of the patients was only slightly less than that of the controls (reclining, 111.5 : 114.9; standing, 118.0 : 120.9). On the other hand, the reactivity of the patients as shown by the change in blood pressure with change from the reclining to the standing posture averaged proportionately considerably less than that of the normal subjects (3.3 : 5.5). In the latter respects, too, the variability among the patients was markedly greater than among the controls, as was shown by the much greater spread between extreme values.

COMMENT

The most important consideration in the attempt to evaluate the findings presented is whether the scoring which was evolved for normal subjects is valid for schizophrenic patients. In other words, are there internal compensating factors that result in a factitious normality of score? Bauer [2a] and Scott [2b] pointed out that an initial low pulse rate loads the score in favor of the subjects. As indicated in table 3, our patients had an average resting pulse rate 5 per cent below that of the controls. If the relationship between bradycardia and raising of the score were linear this would elevate the score about 0.6 point. Correcting our mean score for schizophrenic subjects by this amount would change the ratio of the patient : normal scores to 11.3 : 13.4. Thus the controls would rate approximately 19 per cent higher than the patients. In consideration of the fact that exercise tends to raise the score and that our patients in general led somewhat active lives, this difference is probably significant, indicating a reduction of fundamental physiologic fitness as characteristic of schizophrenia.

8. Hoskins, R. G., and Sleeper, F. H.: Organic Functions in Schizophrenia, Arch. Neurol. & Psychiat. **30**:123 (July) 1933.

A small amount of restlessness of the patients at the time of the test and the distractions in the ward would seem to have caused the scores occasionally to be lower than they should be. But this lowering was probably compensated, if not overcompensated, for by an increase in the score incident to the fairly frequent delay of two or three seconds in obtaining the pulse rate after exercise, the occasional lack of vigor in exercising and the fact that in 33 tests the possible occurrence of a second increase in the pulse rate after exercise was overlooked by following the pulse rate only until it had decreased to or below the standing rate.

It is to be noted that the scores obtained for the group as a whole tend to be higher than those obtained by other observers. Possible explanations for the disparity may be: (1) application of Schneider's later suggestions regarding the technic of the test of which other observers do not seem to have been aware; (2) ruling out tests on patients who had had physical illness near the time of the test; (3) the relatively active physical life led by our patients; (4) the high percentage of active and laboring workers in the group tested; (5) a less disturbed and more cooperative condition of our patients, and (6) the habituation of our patients to testing, a factor that presumably accentuated the initial slowness of pulse rate which tends to elevate the score.

The score of subtypes in our group tended, as to means, to be similar to Reed's, whereas the markedly lower scores of catatonic and hebephrenic patients, noted by Trentzsch, were not apparent. Is there some differential factor in the environmental situation at the Central Islip and the Worcester State Hospitals as compared with St. Elizabeth's Hospital, the treatment tending to keep the hebephrenic and catatonic patients more active? Trentzsch stated that his catatonic and hebephrenic patients showed a purely regressive type of psychosis (which is associated with underactivity), while paranoid and manic patients showed a compensatory projecting type of psychosis (which is associated with normal activity or hyperactivity). The hebephrenic and catatonic patients in our hospital tend to be converted from a purely underactive, regressive type to something of an active type, and Reed stated that his active patients—apparently including the catatonic and hebephrenic subjects—were adequately exercised at farm work, etc. Trentzsch made no comment on the habits of activity of his patients. It is possible, then, that the low scores he reported mirror only the degree of habitual physical activity rather than indicate a characteristic of schizophrenia per se. Actually, the reports from the three hospitals cannot be satisfactorily compared because of inadequate reporting of the concomitant details of the researches.

The analysis of the indexes for the different ward grades in our series brought out only slight differences, but these differences were in the direction one would expect, higher grades being associated with higher Schneider scores and lower grades with lower scores. Since the higher grades represent an approach to or toward normal, it is possible that the differences among the various grades were minimized by the considerations adduced in the first paragraph of this comment.

The individual data—and especially the systolic blood pressure findings—in this study serve to illustrate an important difference between schizophrenic and normal subjects. This same group of patients was elaborately studied under basal conditions and found to have a systolic pressure that averaged 100 mm. of mercury or slightly less.[9] Yet under the slight change of situation involved in testing after a rest period in the afternoon the average pressure was increased to 119 mm.—essentially the same as that of the normal subjects. It is, no doubt, this deceptive ability of the schizophrenic person to rise to temporary normality under stimulation that has often caused the fundamental physiologic abnormality to fail of detection. This topic has been discussed at greater length elsewhere.[10]

SUMMARY

The Schneider test that is alleged to measure cardiovascular fitness was administered to 25 members of the hospital staff and to 99 schizophrenic patients.

The mean score of the patients was 11.9 and of the controls, 13.4—a difference two and one-half times its standard error. Arbitrarily correcting the score of the patients in relation to the initial lower pulse rate would reduce their score to 11.3, a value 19 per cent below that of the controls.

The pulse rates and the systolic blood pressures were slightly lower in the patients than in the controls, but the ranges among individuals were notably greater, as were also the Schneider scores.

The different subtypes of schizophrenia showed substantially equal scores.

CONCLUSION

The Schneider score indicates a significant lowering of physiologic fitness in the schizophrenic patient, but apparently mirrors the degree of physical activity rather more than the intrinsic degree of normality.

9. Freeman, H.: Effect of Habituation on Blood Pressure in Schizophrenia, Arch. Neurol. & Psychiat. **29**:139 (Jan.) 1933.

10. Hoskins, R. G., and Jellinek, E. M.: The Schizophrenic Personality with Special Regard to Psychologic and Organic Concomitants, Proc. A. Research Nerv. & Ment. Dis. 1934, to be published.

MENTAL STATE OF THE EPILEPTIC PATIENT

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The development in epileptic patients of certain traits of character and types of behavior has been so striking that a group of psychiatrists look on the "epileptic personality" as the essential part of the disease. They have used it not only to account for the inability of the epileptic patient to adjust normally to society but even to explain the seizures themselves, which, to their minds, represent "simply a regressive and protective mechanism resorted to by an overstressed organism." [1] Other psychiatrists and the majority of internists regard the peculiarities of the epileptic patient as reactions to environmental restrictions and prejudices, without taking recourse to "hereditary taint" or "constitutional make-up."

Such differences in point of view are of more than academic interest. To believe that the disease is a fundamental defect in personality, hereditary in nature, is to make all therapy palliative. On the other hand, if the affliction is looked on as a disturbance of physiology, and the mental state as secondary, therapy has hopeful aspects, and relief from the disease might be expected to follow closely the growth of knowledge concerning physiologic processes in the central nervous system.

As ordinarily described, the epileptic patient is a self-centered person, constantly demanding his own way and violently resenting interference. He is subject to outbursts of temper and is often moody and peevish. Pugnacity and even unfeeling cruelty may be a part of his make-up. Emotionally he is unstable and extremely sensitive about his own difficulties, but is without feeling or consideration for those of others. He is sluggish in body and in mind, but is sometimes capable of good mental performance if given adequate time. Conversation is dull and monotonously confined to a few subjects. Only with greatest effort are habits of life and thought altered. Of course, not all these traits are exhibited by every person with epilepsy; some are more prominent in one person, others in another, or they may be entirely absent. But considering epileptic patients as a group, one gains the impression

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1. Clark, L. Pierce: Epilepsy and the Convulsive State (Series of Research Publications, vol. 7, 1922-1929), Baltimore, Williams & Wilkins Company, 1931, p 74.

that disturbances in behavior of a similar nature are present with sufficient frequency to warrant the suspicion that in some way they are bound up in the very nature of the disease.

Discussions and arguments over the significance of the epileptic personality have been innumerable, but few studies have been reported in sufficient detail to permit an unprejudiced evaluation of the problem. The opportunity to observe the development or regression of this epileptic personality is never offered by institutional patients, for they are already far advanced in life and disease. A nearly ideal opportunity for such a study would be the experiences of a general practitioner who had known his patients intimately over a period of many years. Observations made under such circumstances have been recorded occasionally in the literature as, for example, by Turner,[2] who stated that he had observed the development of the typical characteristics following episodes of epileptic seizures and had also seen their complete disappearance as a result of successful therapy or spontaneous cure. Diethelm[3] also noted interesting relationships between features of personality and the disease as a whole. In this study two sets of cases have been available which in many ways seem unusually suitable sources of information. The first set comprises several children who were so well known from the social and medical points of view during the course of development of typical epileptic personalities that they afforded unusual opportunities for attempting an evaluation of the factors involved. The second set was a small series of children in whom therapy of one form or another proved so successful that a comparison of the mental state at the height of the disease with that during a remission became possible. The following discussion is presented, therefore, because it helps to clarify the relationship of the mental state and personality of the patient to the disease as a whole.

SITUATIONAL FACTORS

In order to gain insight into the origin of the peculiarities of temperament of the epileptic patient one must know the family background and social setting in sufficient detail to be able to make distinctions between cause and effect. But any one who attempts to do this lays himself open to criticism, for few people will agree as to how completely hereditary influences can be separated from the intricacies of environment. However, in the following case it is felt that some degree of separation and distinction is possible, for the personality unfolded before my eyes.

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2. Turner, W. A.: J. Neurol. & Psychopath. **7**:193, 1927.

3. Diethelm, O.: Epileptic Convulsions and the Personality Setting, Arch. Neurol. & Psychiat. **31**:755 (April) 1934.

CASE 1.—E. was the fifth child in a family of six. The first was living and well, a heavily built, phlegmatic girl; the second died at 15 months after a severe febrile illness which lasted a week and terminated with coma and convulsions; the third child was well; the fourth had "spasms" (colic?) while teething, but otherwise was strong and healthy; the sixth died at the age of 2 months of summer diarrhea. The first bore strong resemblance, both physically and temperamentally, to the father; the other three living children inherited the more slender build and untiring energy of the mother. The father was a faithful, industrious laborer (ship-rigger), completely illiterate but fond of his children. He used alcohol, but not to excess. The mother came from a family which was intellectually superior to that of the father, but one which had shown social instability in recent generations (illegitimate children, addiction to drugs). The mother had great energy and capability. In contrast to the father, who was not of a worrying disposition, she was quick and impetuous, with a temper which flared up on occasions, but was usually kept under control. The mother and one sister were subject to frequent severe headaches of a type which might be classified as migraine.

After a rather long and hard, but otherwise normal labor, the patient, when born, seemed to be normal in all respects and weighed 7 pounds (3,125 Gm.). The father was so bitterly disappointed over the birth of a fourth girl that he left home and did not return for two weeks. He resolved to treat the child as a boy. She grew well on artificial feedings. Development seemed entirely normal, and in some respects even more rapid than usual. At 10 days of age the child had a "spasm," with mild twitchings of the left side of the body. Similar seizures recurred every one to four weeks, but were sufficiently mild not to occasion any particular alarm. At 18 months the child had a violent left-sided convulsion which lasted for eight hours, and was finally relieved by the administration of chloroform. From the age of 3 years, mild to severe convulsions, usually left-sided, occurred every one to three weeks, sometimes as frequently as ten in a day. Various forms of medicinal and dietary therapy were without effect on the course of the disease. These bare facts are necessary to keep in mind as a background.

The patient's first three years differed little from those of other children in the neighborhood. Her occasional "spasms" were not considered serious, for "many children have spasms and outgrow them." From infancy the child possessed an almost untiring amount of physical energy, needed occupation constantly to keep her out of mischief and always enjoyed to the full the tomboy activities which her father encouraged. She frequently wandered into adjoining streets, and at times her mother resorted to tying her to a post with a long cord. This always occasioned violent outbrusts of screaming, which sometimes lasted as long as an hour. The mother, however, would remain firm, and soon E. would go about her simple play. During this period the maternal discipline was possibly somewhat erratic, but on the whole firm and just. But being the youngest of the family and father's favorite, as well as the pet of many friends and relatives, E. was overindulged with all manner of attentions and gifts. Discipline was inconsistently applied because of the conflict between the mother's attempt at sternness and the father's indulgence. At an early age E. learned to demand her own way and, by appealing to the right people in the right way, usually succeeded in obtaining it.

At the time E. was somewhat over 3 the mother accepted steady employment as a means of increasing the family income. The child was left in the charge

of an aunt whose former difficulties with lovers, husbands, and narcotics are indications of her general instability. Also living in the house were the three older sisters, a grandmother and grandfather, several other relatives and the parents. Although fond of the children, the aunt's care and training left much to be desired. Discipline became more than ever a matter of discordant commands, unfulfilled threats resulting in spats back and forth, lengthy arguments, entreaties and, at night when the parents returned, perhaps severe punishment. The result was an increasing disrespect for all discipline. Behavior problems came to the fore in a short time in the way of stubbornness, temper tantrums, resentment and pugnacity. Threats of being "put away" in an institution because of her behavior and "spells" were made frequently during this period, and already the children of the neighborhood had begun to call her "fits" and "crazy." The growing consciousness of her epileptic seizures, which were beginning to assume more serious proportions, offered an excuse to both child and adults for further indulgence and less rigid discipline. But, although creating an increasing behavior problem, she was still the unusually bright, receptive and interested tomboy that made her a family favorite. At the age of 5 years and 10 months she was first brought to the hospital for observation, and was described by a psychiatrist as "irritable, interested, active, imaginative (makes up stories on spur of moment), disobedient." Her behavior was characterized as "quiet, interested, sits quietly in the chair, no twitching or involuntary movements. Stream of talk, answers questions politely and to the point, interrupts rarely." According to the Binet-Simon examination at that time, she was able to do the tests of the 7 year group, which gave a calculated intelligence quotient of 120, and indicated that she had acquired general childhood knowledge somewhat more rapidly than the average child.

E. started to school at the age of 6, but because of her seizures was allowed to stay only two weeks. From that time on she became more and more a subject of nicknames and ridicule by the neighboring children. Although sensitive to their remarks her physical prowess enabled her to fight and force a certain amount of respect. The following episode, which she related with a great deal of feeling, is illuminating. Six or eight children of her age or older were playing together at a game of ball. E. came along and asked to join. One child said, "I won't play if 'fits' plays." Another agreed, "If 'fits' plays I won't play either." A third agreed. Another said, "Well, I don't see why we shouldn't let her play; she can't help it if she does have fits." The end-result was that no game was played, but a general battle ensued in which E. succeeded in driving the others away. Such episodes occurred not infrequently, multiplying antagonism. Quarrels and fights between the children were so frequently carried home to the parents that feuds of almost clannish intensity developed, until the neighbors began to demand that she be "put away."

At the age of 8 all children of her age were attending school, leaving her only younger children to play with. Such circumstances gave full freedom to her egocentric desires and permitted her to be "master of all she surveyed." Nor were the language and practices which she taught the others of such a nature as to make her a desirable playmate. More quarrels and fights arose, finally reaching a climax when several neighbors had the patient brought into juvenile court with the demand that she be "put away." She was charged with picking quarrels with other children, especially little ones, keeping the neighborhood in a constant uproar, teaching the children sex practices and telling obscene stories. However, the case was dismissed with the admonition to the parents to keep the child at home. The experience has never been forgotten

by the patient, who still looks on it with remorse and fear. It has seemed to make her even more sensitive to criticism and to heighten her jealousy and antagonism.

At the age of 8, while under observation in the hospital wards, E. offered no particular problems of behavior which were not easily understood on the basis of her extremely active nature and limited outlets. Perhaps one reason was that she remained under the care of a special nurse in a room by herself, with little opportunity for contacts with other children and for demonstration of her social antagonism. She appeared friendly and unreserved, not particularly introspective or oversensitive about her seizures, was easily interested in any number of things and cooperated well with examinations and treatments. Intellectually, she gave the impression of having at least average ability, although with her almost restless activity the span of attention may have been somewhat reduced.

She was admitted to the hospital again at the age of 10, under conditions that were as ideal as could be expected in the routine of hospital life. She was not kept in bed, but had the freedom of two rooms and a large sun parlor, with periods of an hour or two in which she could be out in the hospital grounds playing ball and croquet, watching games of tennis and taking long walks. Three or four other convalescent children of about her age were available for playmates. They all were good natured, well brought up children and gladly accepted her as one of the group. An attendant, thoroughly conversant with the ways of children, was constantly present. Hopes were entertained of accomplishing some degree of socialization and of teaching unselfish play.

During a short period of "Sunday behavior" all went well. She, however, constantly demanded her own way. Gradually her egocentricity began to assert itself more and more; she demanded her own way with increasing firmness until, with little provocation, long periods of pouting occurred and finally violent temper tantrums lasting as long as an hour. If corrected for some misdemeanor she seemed to take delight in repeating the performance whenever the opportunity offered itself. Little quarrels between the children which necessitated a decision in favor of one of the others provoked violent jealousy and retribution. On one occasion after such an episode she stuck her finger into a healing osteomyelitis wound of a playmate. Another time she struck a child violently for no obvious reason except that she herself had recently been punished. The barest suggestion of discrimination or partiality was magnified hundreds of times in her eyes. But in spite of this, during periods of good behavior she gave evidence of a certain amount of understanding and remorse by such remarks as "Have I been good today?" Periods of jealousy, pouting and temper tantrums were increasingly frequent, until it became impossible to keep her longer in the ward.

During the stay in the hospital she took considerable interest in school work, assisted by the nurse and a visiting teacher. Memorizing a reading lesson (first and second grade) proved easier than learning to read it. Arithmetic was somewhat difficult because of the failure of application, but also from disinterest and difficulty of retention. At times she would pretend to study diligently, but as soon as the attendant seemed satisfied she would burst into a roar of laughter to see how complete the deception had been. Obviously, a great change had occurred since her first visit to the hospital three years previously. She now exhibited in high degree the cardinal symptoms of the so-called epileptic personality—egocentricity, supersensitiveness, emotional poverty and rigidity of ideation and mentation. Observations were also made which might well indicate mental retardation; how correct such an interpretation may be is not easy to judge.

Even at the time of the first admission E.'s knowledge of moving picture actors and actresses, love-making and matters pertaining to sex was advanced, and occupied a prominent place among her interests. However, no special attention was paid to the matter, for it was known that, like her sisters, she had accorded movies a regular and predominant place among her amusements. Her interest and knowledge in these respects were simply an indication of the family life. During the four years of her association with the hosiptal this interest in sex matters continued to grow. As already mentioned, one of the complaints to the juvenile court pertained to sex practices. The attempt at home to suppress rigidly all information on such matters seems, however, to have encouraged her interest.

Since early childhood E. had taken a more serious interest in church and Sunday-school than her three older sisters. When, during adolescence, the others drifted away from church, her interest remained, although opportunities of attendance were few. The Salvation Army began to hold open-air meetings near her home on Sunday night, and immediately she became a regular attendant. Her loud voice and love of singing, with the occasional epileptic seizure in public, soon brought her to the attention of the leader, who encouraged her to sing as one of the choir and moved the place of their meetings nearer to her home.

When the patient was 10½ years of age the family moved into a different locality, in which the neighbors were friendly and the children less antagonistic. None of the relatives now lived with the family. Because of financial difficulties indulgences became less, but partiality and an inconsistent and conflicting discipline continued. Gradually E. became less difficult to get along with. Quarrels and battles still occurred with other children, but lasted only a short time and were frequently forgotten the following day. Rarely did they occur over epithets of "fits" and "crazy," for E. was gradually realizing that combat did not counter such charges. A visiting school teacher came regularly and succeeded in maintaining a lively interest in school work. At this time both teacher and pupil recognized a difficulty in remembering facts, which was particularly noticeable for a few days after a series of convulsions. Arithmetic lessons were done with greater ease than reading. E. was ready to do "anything to learn," in the hope of being able some day to earn a living and care for herself. Toward the housework, also, she took a more helpful attitude, regularly doing a part of the family washing and ironing. Her disease was now looked on in a fatalistic way and, although she believed that she would "never get over it," she blamed no one in particular. However, she still gave long accounts of injustices done her by family, friends and playmates, all of whom still seemed to her to be set against her. Love and sex were frequent subjects of conversation. Generally speaking, her character did not differ greatly from that of preceding years, but her reactions were of a more mature kind in contrast to her former childish ways.

Comment.—Of course, in the life of every normal person environmental influences constitute an important and fundamental part of the molding process which finally results in the developed personality and individuality. Whether protracted illness produces a sense of invalidism or an attitude of "the devil may care," the response depends on the individual and on the wall of social contacts which surround him. In the case of the person suffering from epilepsy this social wall frequently becomes an insurmountable barrier to all the usual outlets of human interests. Such circumstances can hardly fail to leave their marks on the character of the person with epilepsy and in this patient have been, without doubt, a factor of major importance in the development of the peculiarities of disposition. In this case the endowments of aggressiveness and robust physical structure have probably contributed to the type of response elicited.

The foregoing presentation is but a single example of the importance of environmental influences on the development of the peculiarities of epileptic patients. If time and space permitted, accounts might be presented of patients with epilepsy and typical personalities whose disease remained little influenced by therapy, but whose psychologic state was so altered that no one who was not intimately familiar with the previous history could have detected evidences of the epileptic personality. Thus, if the personality can be normalized without affecting the disease, the former can hardly be called a cause of the latter. The task of making such an adjustment is far more difficult than the administration of drugs and diets, and few physicians are interested in or capable of doing this type of work; consequently, the examples are not numerous. However, they are sufficiently common to point out once more that the personality features are not an essential part of the disease.

PHYSIOLOGIC FACTORS

In addition to the tremendous influence of the home and social life in creating the common peculiarities of epileptic patients, other factors are operating which are a part of the disease itself. Extreme fluctuations in the mental state often closely parallel the seizures. This and the fact that certain mental symptoms completely disappear during a remission in seizures justify the view that temporary functional disturbances constitute some part of the clinical picture, and unless such a possibility is recognized a single examination of a patient may give a false impression.

Frequently, it is not a simple matter to distinguish the effects of the social setting from the symptoms which depend on the disease itself. Seeming mental retardation, monotony of interests and conversation, dulness of mind and sluggishness of physical activities are symptoms seen not uncommonly; in other patients, sometimes alternating with the aforementioned are flaming color, brilliant eyes, peevishness, irritability, overactivity and destructiveness. Both sets of symptoms are usually regarded by members of the medical profession as representing phases of the disease from which little improvement can be expected and denoting that the disease has progressed to such a point that mental deterioration, institutional care and death are to be anticipated. Such a course of events is by no means inevitable. In fact, the opposite occurs with sufficient frequency to suggest that the present use of the mental state as an indication of prognosis may be unfounded, and that the same disturbances of physiology which give rise to the seizures affect the entire functioning of the brain in such a way as to produce the forms of mental symptoms described.

The following case reports are offered as illustrations:

Case 2.—A boy, aged 8 years, began to have major convulsions at the age of 4. Except for a period of eighteen months of complete freedom from seizures, ending six months before he came under observation, severe generalized convulsions had occurred from two to four times a week. Medication with phenobarbital had been irregular and unsatisfactory. When first seen he was severely defective mentally with a dull, vacant expression and an open and constantly drooling mouth and in an unending state of overactivity, never satisfied with anything for more than a few moments. He had to be watched constantly to prevent him from doing undesirable things and from being destructive. Because of certain physiologic factors he was given a drug heretofore unused in epilepsy.[4] Two weeks later the mother reported slight improvement. A month after the beginning of the treatment the child had had no seizures, and the mother offered the information that he seemed more quiet, attentive and agreeable. The boy did not return to the clinic for a year because, as the mother said, he seemed so well that she saw no need of his returning. Not a single epileptic seizure had occurred during this period. The mother reported that during the year she, herself, had gained 20 pounds (9.1 Kg.), which she attributed to the fact that she had had little work to do in caring for her son since his improvement. On examination he still appeared to be a very defective child, but in contrast to the previous year now sat quietly in a chair, playing with simple toys for long periods of time, answered questions and talked freely, quietly and openly; he was not restless or overactive, and saliva rarely dripped from his mouth. The mother stated that likewise at home he would sit quietly for hours playing by himself and listening to stories. The change in his behavior was striking.

This case helps to distinguish the several components which contribute to the clinical picture of the mental state. Although no "intelligence tests" were utilized, it is not to be expected that the child's basic intellectual level would change appreciably as a result of treatment. The peculiarities of disposition and behavior, which made this boy so disagreeable, unmanageable and a social outcast completely disappeared along with the seizures as soon as effective medication was instituted. This phase of the mental picture can be interpreted only as one more indication of the activity of the underlying process and not as necessarily suggestive of a poor prognosis. Thus, at least three important factors are contributing to the clinical picture of the mental state: (1) stimuli of an adverse environment; (2) anatomic damage to the brain, and (3) a functional or physiologic disturbance, which appears to be linked in some way with the activity of the disease process.

Case 3.—A boy, first seen at the age of 5 years, had epilepsy first at the age of 4 without apparent cause. Major convulsions occurred every two to four weeks, and recently petit mal seizures several times a day. Phenobarbital in doses

4. Although clinical improvement coincided almost exactly with the administration of the drug (theobromine sodium salicylate) in this case, the result has not been duplicated in other cases. However, the clinical transformation, and not the mechanism behind it, is pertinent to the present discussion.

of ½ grain (0.025 Gm.). three times a day had been without effect. When first seen the boy was normally intelligent, active in his play and never able to sit quietly. It was difficult to keep his attention on any one thing for more than a few minutes. While in the clinic he ran with tremendous energy up and down the hall. His cheeks were intensely flushed, his eyes bright and shining. The mother had observed that during the periods in which seizures were most frequent the overactivity and flushing of the face were even more intense than at other times. He slept restlessly and for comparatively short periods.

He proved unusually amenable to medical treatment, and for three years has been without seizures. Concomitant with the disappearance of the attacks the overactivity vanished, the intense flushing of the face subsided and he slept more quietly and was comparatively easy to manage. At present he is in all respects a normal boy, standing among the first few in his class at school. In this case certainly one should not have offered a poor prognosis on the basis of his peculiarities of temperament alone, which, in the light of the present information, must have been purely functional.

Case 4.—A girl, aged 7 years, who previous to the present illness had been healthy, did second grade school work without difficulty. Brief petit mal seizures began to occur for no apparent reason. They were infrequent at first, but after a few weeks came with increasing frequency and severity, until four months after the onset moderately severe convulsions were experienced several times a day. Within about two weeks of the onset difficulty in speech was noticed. This progressed steadily, in a general way parallel to the severity of the seizures. No regular medication had been employed. When seen, the child could say only a few words. At that time she was irritable and restless, wandering constantly from one thing to another. She could still write a few words that had been learned in school and could perform simple first grade arithmetic tests. A Binet-Simon test of intellectual capacity was obviously impossible under the circumstances. A diagnosis of tumor was made, but an injection of air into the ventricles showed no abnormalities. Medical treatment was begun, and improvement in the seizures and in the mentality was noted almost immediately. Within four months the child talked with only slight difficulty, and her general activities were fairly normal. Eight months later she would have passed as a normal child to any one who was not on the lookout for her few remaining difficulties in speech.

Case 4 is cited as an example of extreme mental and psychologic changes which seemed to parallel closely the severity of the epileptic seizures but, as judged by the outcome, must have represented functional rather than structural changes in the brain.

Case 5.—A girl, beyond the age of puberty when first seen, had had convulsions at irregular intervals since the age of 9 years. Frequent daily petit mal seizures began several years later. In spite of constant medication with phenobarbital and bromides the symptoms of the disease went through wide fluctuations; menstruation and psychogenic factors, among others, seemed to aggravate the condition. During periods in which she was free from seizures the mind was alert, the eye bright, the attention good and conversation wholesome and entertaining. A week later, when petit mal seizures were numerous, the entire picture might be different. She seemed dull and was listless; her conversation was constant and monotonous, sometimes maintained on a single theme for as long as an hour at a time. Sex topics were frequently the subject of these monotonous discourses; repetitions were frequent. With great difficulty the subject could be changed, only to revert in a short

time to the previous topic. A few days later another change might occur which would make her a pleasant and apparently normal person. Such fluctuations in the mental state can in no way be interpreted as evidence of organic deterioration of the brain; they probably are effects of the same underlying disturbances that give rise to the epileptic seizures.[5]

Instances such as these are by no means unusual in a group of persons with epilepsy. In all probability the phenomenon occurs to a greater or lesser degree in all patients with the disease. Consequently, in evaluating the mental state of any epileptic patient the functional aspects cannot be disregarded, for by so doing one may be led far astray from the correct diagnosis and prognosis.

ANATOMIC FACTORS

There are certain structural changes in the brain of epileptic patients which must contribute in some degree to the mental state. Gross abnormalities of the brain as a background of repeated convulsions are seen frequently. The association of epilepsy with cerebral tumors, scars, spastic paraplegia and congenital malformations of the brain is common. Under such conditions one naturally could not expect the mental functions of the patient to be left intact. All gradations from slightly impaired intelligence to imbecility are seen in such a group of patients. Certainly in any evaluation of the mentality of an epileptic patient the degree of the gross defect of the brain must be considered; and from the increasingly frequent demonstration of such abnormalities this factor must be of more significance than has been considered heretofore.

The anatomic basis for the wide variations in mental deterioration among epileptic patients is a matter of much dispute. In some patients

5. It is difficult to explain such phenomena with the present understanding of the physiology of the brain and in ignorance of the factors involved in the production of convulsions. But reasoning from analogy one can at least form a mental picture of the course of events which, whether correct or not, offers a more helpful point of view. An increasing amount of evidence is linking convulsions and epilepsy with disturbances of the autonomic nervous system; this evidence suggests that the convulsions represent the response of the autonomic elements of the brain to impulses which may arise anywhere throughout the system. Vicious circles become involved as the disease progresses. Vasomotor phenomena constitute a part of such a picture and undoubtedly contribute to the symptoms and course of the disease. In the light of the mental symptoms in cerebral arteriosclerosis, severe anemia, cardiac failure, polycythemia and states of reduced oxygen tension, it seems not unlikely that many of the changing mental symptoms in epilepsy might be explained as fluctuations of a borderline or slightly insufficient nutrition of the brain. The degenerative changes in the brain, considered by Spielmeyer [8] to be of vascular origin, might be a late result of such a state and of the repeated severe cyanosis of each convulsion. Such a conception is, of course, largely speculative, but it offers a hypothesis which not only agrees with many experimental observations, but offers a basis for understanding the individual patient and his treatment.

marked mental changes develop within a year or two in association with increase in the epileptic seizures; in the majority of patients deterioration is not apparent even after many years.[6] These facts suggest that the mechanism which produces the changes is by no means always the same. No doubt there exist within the group of patients now labeled epileptic, distinct disease entities which have a variety of anatomic lesions. Examples are to be found in such conditions as progressive cerebral degeneration, encephalitis periaxialis diffusa (Schilder's disease) and lead encephalopathy in children, as well as in tumor of the brain, vascular disease, etc., in adults, all of which may be indistinguishable from epilepsy in their incipiency. Understanding of the nature of mental deterioration will undoubtedly advance as diagnostic methods develop which will enable the physician to distinguish such a group of diseases from the large mass of epilepsies.

The problem would no doubt be greatly clarified if it were possible to distinguish at autopsy between primary and secondary cerebral changes, between the pathologic aspect of the disease process itself and the damage which results from the disease, as, for example, from the convulsions. Many facts seem to support the conception that the intense intracranial pressure developed with each convulsion results in progressive damage to the brain tissue and eventually leads to deterioration. Fay,[7] a recent advocate of this conception, expressed the belief that the dilated subarachnoid spaces with the underlying cortical atrophy, long recognized in association with all forms of convulsive disorders, are an expression of the effects of this increase in pressure and are localized in areas of least resistance. In addition, Spielmeyer [8] described degenerative processes observed histologically which seem to correlate roughly with the severity and duration of the disease. He expressed the belief that they are anoxemic in origin. Possibly an underlying disturbance of the cerebral circulation, as well as the frequently repeated severe cyanosis of each convulsion, may contribute toward this type of degeneration.

THE INTELLIGENCE QUOTIENT

It is commonly believed that patients with epilepsy are, or will eventually be, mentally deficient. While it is true that many epileptic persons suffer impairment of their mental faculties, recent studies have shown that this aspect of the disease has been grossly overemphasized. Lennox's [6] analysis of noninstitutional patients showed that 54 per cent

6. Lennox, William, in Nelson Loose Leaf Medicine, New York, T. Nelson & Sons, 1929, vol. 6, p. 628.

7. Fay, Temple: J. Nerv. & Ment. Dis. **71**:481, 1930.

8. Spielmeyer, Walther: The Anatomic Substratum of the Convulsive State, Arch. Neurol. & Psychiat. **23**:869 (May) 1930.

were normal mentally, even though the disease had existed for more than twenty years.

Until comparatively recently the task of sizing up the mental state of a person has not been guided by fixed rules; the character and disposition were reviewed with respect to the ability to live successfully and harmoniously with associates. The appraisal depended on the experience and intuition of the examiner, and had no objective value. With the advent of standardized intelligence tests, an instrument became available, even to the inexperienced person, which permitted one to perform certain tests of intellectual ability and compare the results with set standards.

While popularization of studies of the intelligence quotient has proved a tremendous advance by broadening the scope of usefulness, it has not been without its drawbacks. Errors are easily made if such examinations are used as yardsticks, without consideration of the individual subject and his problem. The sale by modern department stores of equipment and directions for performing these tests—under the caption "How bright is your child?"—cannot fail to produce an additional crop of behavior problems among the coming generation. The very ease with which such examinations can be given has permitted the growth of a generation of "testers" who know little of the background on which such studies are based and who, consequently, can interpret their results only in accordance with fixed rules.

While it seems hardly necessary to point out several of the pitfalls of intelligence tests, for they are well recognized by more progressive psychiatrists, the fact that epileptic patients are so frequently misunderstood is justification for mentioning the subject. In the first place, the tests in common use utilize the abilities of children in the various school grades as standards. No satisfactory comparison can thus be made with epileptic children who have been kept away from school. While certain tests are available for such contingencies, they are not usually employed, and the epileptic child is thus at a decided disadvantage. Again, a general slowness of response is often characteristic of epilepsy, and unless this is appreciated the Binet-Simon score may show failures that are due not to low ability but to inadequate time. Possibly the drugs which patients usually receive are conducive to this type of sluggishness. The social handicaps which the disease necessarily imposes almost automatically make the child more sensitive, fearful and apprehensive than otherwise, and, since mental calmness is an asset for any examination, it should be encouraged, especially when epileptic children are being studied. Finally, if it is true, as psychiatrists have suggested, that patients with the disease exhibit certain characteristics more than others, as, for example, an ability to deal with concrete and mathematical subjects in contrast to creative and imaginative ones, such facts are

of importance in evaluating the successes and failures of the tests. Differences in nationality, language, social standards and cooperation are familiar obstacles in Binet-Simon examinations, which need allowance in epileptic and normal children alike.

The failure to recognize such sources of error in the interpretation of the intelligence quotient and an overemphasis on the result obtained has frequently led to grave misunderstanding and injustice to epileptic children. For the patient discussed in case 1, an intelligence test done before school age gave an intelligence quotient of 120; at the age of 10, 84; and at 11½ years, 81. In spite of the fact that the girl had had essentially no schooling and few opportunities for intellectual development at home, the second test was considered to indicate definite mental deterioration, and the child was accordingly recommended to a school for the feebleminded. A broader view of the significance of intelligence quotients would have included the consideration of the influence of the varied situational factors.

The importance of a realization of the limitations of intelligence quotients can hardly be overstressed. For studying statistically a large group of essentially normal children such tests have been of inestimable value as one of numerous ways in which to evaluate an individual, especially a child; as a guide in adjusting him to school and society such examinations are essential. When used as the basis for judging a person whose life and training have been different from the average, grave pitfalls open up, for the standards which are based on school grades as units can no longer be employed. Nor are there available at present any standardized ways of evaluating such qualities as experience, interest, cooperation, persistence and conscientiousness, which, in the last analysis, are of equal importance with intellectual capacity as far as success in life is concerned. In the minds of many workers the modern tests of intelligence can be interpreted only as showing that an individual child has developed more or less than the average children of his age; no light is thrown on the reason for the difference.

SUMMARY AND OUTLOOK

How, then, is one to view the mental state of the epileptic patient? Obviously, instead of being a question simply answered, the problem is as intricate as personality itself. In all probability the so-called epileptic personality is not an entity which bears any specific causal relationship to the disease, but represents, in large part, the response of such patients to the problems and situations which the very nature of their disease creates. In addition to the tremendous influence of environment on the personality of epileptic patients, the underlying process has an undoubted effect on the mental state. The interference with normal

cerebral function caused by whatever scarring may be present in the brain, the general restlessness, irritability and loss of memory of a temporary functional nature observed during the active and progressive stages of the disease, and, finally, the damaging effects of the convulsions themselves on the nervous system, are all of unquestionable importance in producing the clinical picture of the epileptic personality, psychosis and dementia.

A recognition of the nature and significance of the mental state of the epileptic person has far-reaching implications. In the first place, it should be recognized that there exists a group of patients, formerly classed as suffering from idiopathic epilepsy, in whom psychogenic factors are of utmost importance in inducing the abnormal physiologic response of convulsions. In such instances routine treatment with drugs, diets and surgical intervention is of less importance than properly directed adjustment of the problems of the mind. Similarly, time, energy and optimism would be wasted if one attempted to treat solely by mental hygiene the patients in whom the dominant alteration of physiology depended on local scars of the brain, abnormalities of the cerebrovascular supply, disturbances of the formation and drainage of the spinal fluid, repeated bouts of fever and tetany. Only as one is able correctly to evaluate the various disturbances of function which in a given person bring about the symptoms now spoken of as epilepsy can therapy be direct, specific and fruitful.

In the second place, the popular conception of an inherited deteriorating personality as the underlying cause of epilepsy has resulted in great misunderstanding and injustice both to the patient and to his family. The fear, prejudice and even aversion which a person with epilepsy so often encounters when seeking education, employment or entrance into social activities can be attributed in considerable measure to such an attitude toward the disease. It is not uncommon to encounter families of excellent stock who dread to have the children they desire, because of the possibility of transmitting to them the taint of epilepsy. A family skeleton of epilepsy is a social disgrace. Finally, the treatment which patients with epilepsy receive from physicians is no doubt influenced by the hopelessness which is bound to result when hereditary aspects of the disease and the futility of curative measures occupy as prominent a part in the literature and teaching of epilepsy as they have in the past. The attitude of society toward epilepsy deserves study, for only as a more rational view of the disease is taken by the public in general, and the medical profession in particular, can the epileptic patient receive the therapeutic aid and the opportunity for normal existence which every human being deserves.

FACIAL AND MENINGEAL ANGIOMATOSIS ASSOCIATED WITH CALCIFICATIONS OF THE BRAIN CORTEX

A CLINICAL AND ANATOMOPATHOLOGIC CONTRIBUTION

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In recent years attention has been drawn to a strange picture in the roentgenograms in some cases of suspected angioma of the brain, especially in cases associated with epilepsy. The diagnosis of angioma of the brain has been made in these cases because of the presence of an angioma of the skin of the face. None of the patients has died, so that no autopsy has hitherto been performed.

In 1921, Ove Wissing demonstrated before the Radiological Society of Copenhagen a roentgenogram of the skull of a young man which showed an unusual shadow. This shadow, which was localized in the right occipital lobe, had a sinuous form corresponding to the outline of the pia mater. Consequently, it was considered to be a calcification of the pia mater. This report was not published, as the Radiological Society of Copenhagen had then only recently been established.

The first published case was that of Parkes Weber [1] (August 1922). The patient, a woman, aged 22, was obese and somewhat infantile and imbecile and had a congenital right hemiplegia and hemihypotrophy; she had also extensive vascular nevi. Roentgen examination showed that the left cerebral hemisphere was less transparent than the right and seemed to be sclerotic. Unfortunately, in the reproductions of the roentgenogram it was impossible to recognize the folded form of the shadow. Parkes Weber, however, in 1929, published a new description of the case, in which the sinuous shadow corresponding to the brain surface was shown clearly.

In 1923, Dimitri [2] presented before the Neurological Society of Argentina a case in which an angioma of the brain showed a distinct shadow in the roentgenogram.

From the Department of Neurology, Kommunehospitalet.

1. Weber, F. Parkes: Right-Sided Hemi-Hypotrophy Resulting from Right-Sided Congenital Spastic Hemiplegia, with a Morbid Condition of the Left Side of the Brain Revealed by Radiograms, J. Neurol. & Psychopath. **3**:134, 1922; A Note on the Association of Extensive Hæmangiomatous Nævus of the Skin with Cerebral (Meningeal) Hæmangioma, Especially Cases of Facial Vascular Nævus with Contralateral Hemiplegia, Proc. Roy. Soc. Med. (Sect. Neurol.) **22**:431, 1929.

2. Dimitri, V.: Rev. Asoc. méd. argent. **36**:1029, 1923.

Marque,[3] in 1927, published a description of two cases. The cases were those of two children who suffered from epileptic seizures and had angiomas of the face. Roentgenograms of the skulls showed folded shadows corresponding with the surface of the cerebral hemispheres in the region of the occipital lobes.

Brushfield and Wyatt,[4] in 1927, reported a similar case in a girl, aged 11, with hemiplegia, imbecility and extensive vascular nevi. Roentgenography revealed shadows corresponding with the brain surface.

Vincent and Heuyer [5] presented before the Neurological Society of Paris, in February 1929, two cases of cerebral venous angiomas which gave rise to shadows in roentgenograms. The first case was that of a boy, aged 4 years, who had epileptic attacks and presented a vascular nevus on the right side of the face. Roentgenograms presented a sinuous shadow in the right occipital lobe. The second case was that of a boy, aged 13, who had epileptic fits, which were at first generalized and later of jacksonian type; in addition, he had a right hemiplegia with contracture. The left side of the face was the site of a vascular nevus. A roentgenogram of the left side of the skull presented a sinuous shadow (unfortunately, the pictures are reproduced so indistinctly that the form of the shadow is not recognizable).

Laignel-Lavastine, Delherm and Fouquet,[6] a month later, presented before the same society a patient, aged 28, suffering from the same disease.

Moore,[7] in 1929, described the following case. A boy, aged 12, complained of defective vision of the left eye. There was a nevus of the left half of the face, which involved the eyelids. He had also right homonymous hemianopia. A roentgenogram revealed a large wedge-shaped shadow (which Moore considered to be caused by a tumor). It was an angioma, consisting of large vessels, with its base at the occipital lobe.

L. Cavel,[8] in 1931, wrote a thesis on calcified angiomas of the meninges. He reported the case of a girl, aged 12, who had had from

3. Marque, Alberto: Consideraciones sobre angiomas en la infancia, Rev. oto-neuro-oftal. **1**:202, 1927.

4. Brushfield, T., and Wyatt, W.: Hemiplegia Associated with Extensive Nævus and Mental Defect, Brit. J. Child. Dis. **24**:98 and 209, 1927; **25**:96, 1928.

5. Vincent, Clovis, and Heuyer, G.: Présentation de deux cas d'angiome veineux cérébral, Rev. neurol. **36**:233 and 509, 1929.

6. Laignel-Lavastine, Delherm and Fouquet: Epilepsie jacksonienne par angiome cérébral avec nævus frontal (présentation du malade et de stéréoradiographies), Rev. neurol. **1**:475, 1929.

7. Moore, R. F.: Haemangioma of Meninges Involving Visual Cortex, Brit. J. Ophth. **13**:252, 1929.

8. Cavel, Lucien: L'angiome calcifié des méninges, Thèse de Paris, 1931.

birth a nevus of the right frontal region. She was mentally retarded but seems not to have suffered from epilepsy. A roentgenogram of the skull showed what Cavel called a calcified angioma of the meninges; in the right parietal region was a mass which was crossed by sinuous lines. The article is not illustrated, but it seems probable that this case presented the same sinuous shadow as the cases previously described.

Péhu, Déchaume and Boucomont,[9] in 1932, published a report of a case of infantile epilepsy with intracranial calcifications. They considered that the shadows in a roentgenogram indicated calcified angiomas of the meninges. However, the description of the roentgenogram does not correspond exactly with that of so-called calcified angiomas. It seems rather to be composed of multiple calcifications, disseminated both deeply and superficially in the brain substance. The patient had had epileptic seizures since the age of 5 years. The right side of the body was paretic for from eight to fifteen days after each fit. An oblong angioma was present over the left eyebrow. Roentgenograms of the skull showed a folded shadow corresponding with the brain surface, which the authors considered to be caused by calcified vessels.

There have also been reported in the literature on brain angiomas several cases with calcifications of the brain or angiomas of the face. During the last few years may be mentioned cases reported by Geyelin and Penfield,[10] Dollfus and Renard,[11] Péhu, Dechaume and Boucomont,[9] Schiele,[12] Schaefer,[13] Baruk[14] and Heuyer and Vogt.[15] It seems probable that most of these cases were instances of angiomas of the brain with calcification. However, the roentgenograms generally

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9. Péhu, Déchaume and Boucomont: Sur un cas d'épilepsie infantile avec calcifications intracraniennes (angiome calcifié des méninges probable), Lyon méd. **64**:249 (Feb. 28) 1932.

10. Geyelin, H. Rawle, and Penfield, Wilder: Cerebral Calcification Epilepsy (Endarteritis Calcificans Cerebri), Arch. Neurol. and Psychiat. **21**:1020 (May) 1929.

11. Dollfus, M. A., and Renard, G.: Un cas de tumeur cérébrale et de calcification méningée visible à la radiographie, sans autre symptôme clinique, chez un acromégalique, Rev. d'oto-neuro-ocul. **4**:603 (Oct.) 1926.

12. Schiele, B. G.: Ueber vorwiegend perivasale sekundär verkalkende Konkrementbildung im Hirnwebe, Virchows Arch. f. path. Anat. **282**:790, 1931.

13. Schaefer, W.: Ueber einen Fall von halbseitigen Gehirn und Hauthämangioma, Monatschr. f. Kinderh. **50**:38, 1931.

14. Baruk, Henri: Migraines d'apparence psychogénique suivies d'épilepsie Jacksonienne dans un cas d'angiome cérébral, Encéphale **26**:4, 1931.

15. Heuyer and Vogt: Crises épileptiques et tumeur cérébrale, vraisemblablement angiomateuse, Arch. de méd. d. enf. **34**:27, 1931. Kalischer, S.: Demonstration des Gehirns eines Kindes mit Telangiectasis der linksseitigen Gesichtskopfhaut und Hirnoberfläche, Berl. klin. Wchnschr **48**:1059, 1897.

have shown massive calcifications rather than the characteristic sinuous shadows corresponding to the brain surface, such as were originally described by Parkes Weber and Dimitri.

Except for these cases, the disease seems to be rare. Cushing and Bailey,[16] in their monograph on angiomatous tumors of the brain, reproduced the picture described by Marque but had not observed any case in their large material.

I have had the opportunity to observe five cases, one of which I have studied histologically. As no such histologic examination seems to have been made before, I shall report the cases here.

REPORT OF CASES

Case 1.—This case (that of H. E. R.) is the one demonstrated by Ove Wissing before the Radiological Society of Copenhagen in 1921. There was no history of a familial predisposition. From the age of 2 years the patient had had epilepsy; at the onset the fits were very frequent, but later they occurred more rarely. After the age of 16, the fits again became more frequent. They occurred always in the daytime, being especially frequent after emotion or after strenuous work. They were of typically epileptic character. There was no aura, but after the seizures the patient was confused for a short time. In addition, he had enuresis nocturna. Except for these symptoms he felt well. He was treated by the administration of phenobarbital for a short time.

In 1921, the patient was admitted to the surgical department C (Professor Rovsing) of the University Hospital of Copenhagen for an operation, but the operation was not performed. Examination at the hospital revealed: general appearance, normal, except for a circular angioma, 3 cm. in diameter, on the forehead above the right eye, limited by the eyebrow; eyegrounds and visual acuity normal on both sides; results of otoscopic and acoustic examination, normal; under vestibular examination, postrotatory nystagmus toward the right; results of neurologic examination, normal. Lumbar puncture was not performed.

The Wassermann reaction was negative. The Pirquet reaction was positive. Roentgen examination revealed (fig. 1): There was a strange shadow, approximately of the size of an egg, in the region of the left occipital lobe. This shadow was sinuous and presented the same form as the brain surface. The sharp lines corresponding to this surface had double contours in some parts. The posterior and inferior parts of the shadow were distinctly limited, but in the anterior part there were worm-shaped prolongations in the temporal region, which did not reach the central convolutions.

In 1929, eight years later, I examined this patient again. A new roentgenogram showed the same shadow as had been observed in 1921; it had not increased in size.

Case 2.—*History.*—E. A., a boy, aged 7 years, who was treated in the "Fuglebakke" hospital for children in Copenhagen (Prof. V. Poulsen) was the youngest of five siblings; the other four had died, one from convulsions and another from pneumonia with a convulsion. A brother of the mother had suffered from epilepsy. The patient was delivered with forceps. The weight at birth was about 3,000

16. Cushing, Harvey, and Bailey, Percival: Tumors Arising from the Blood Vessels of the Brain, Springfield, Ill., Charles C. Thomas, Publisher, 1928.

Gm. The first tooth appeared at the age of 9 months. The child began to speak and walk at the age of 3½ years. The mother had observed that the right foot was dragged somewhat and turned outward.

At the age of 6 years the patient began to suffer from convulsive seizures in the right arm, but he was able to use this hand as well as the left. Sometimes he had fits of weakness of the right leg which caused him to fall. Mental development was somewhat retarded. He had not been admitted to school, but he was able to play like children of his age. In other respects he was well.

Examination.—The child was large and somewhat fat, presenting somewhat the appearance of myxedema. The eyebrows were sparse; the hair of the head was normal. Ophthalmoscopic examination gave normal results. The pupils were

Fig. 1 (case 1).—Roentgenogram of the skull.

equal and reacted to light and in convergence. Nothing abnormal was detected in the cranial nerves. The upper limbs were normal as regards the joints, tonus, trophic condition, power, coordination, sensibility and reflexes. The abdominal reflexes were normal. The joints of the lower extremities were normal, and power was fairly good. The right leg was 2 cm. shorter than the left, and there was some muscular atrophy of the right lower limb. The knee jerks were very active, with a crossed adductor reflex; the right knee jerk could be produced from percussion of the tibia. Plantar stimulation on the right caused a definite extensor response; the response was doubtful on the left. The gait of the right foot was spastic.

Lumbar puncture yielded a clear fluid, containing 21 cells per cubic millimeter, no globulin and a normal amount of albumin (40 mg. per hundred cubic centimeters). The Wassermann reaction of the blood and spinal fluid was negative.

Roentgen examination of the head showed an irregular shadow, about 11 cm. long, in the left parietal region. The shadow was situated immediately beneath the cranial wall; it had the form of a sinuous plate, corresponding with the cerebral surface, but this was not so distinct as in case 1 (fig. 2).

Course.—During his stay at the hospital the boy gave the impression of some oligophrenia; his conduct was strange; he drank his urine when he thought that he was unobserved. He had several clonic seizures in the right arm, during which he seemed to be somewhat excited but was not unconscious.

In November 1928, the boy was treated in the children's department of the University Hospital (Rigshospitalet) of Copenhagen. He continued to have spasms of the right arm and leg, without unconsciousness. The fits had become more frequent, now appearing from two to three times a day. Examination at this time revealed nothing new.

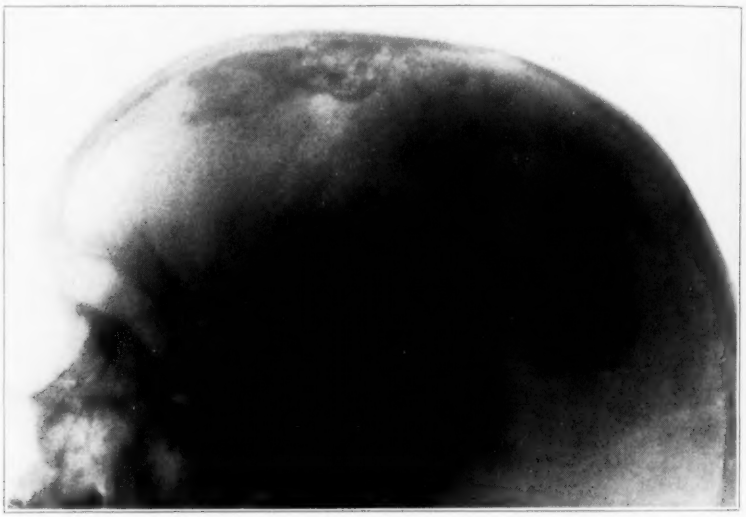

Fig. 2 (case 2).—Roentgenogram of the skull.

CASE 3.—*History.*—G. V. S. was discovered when, after having seen the other patients, I asked Dr. H. I. Schou, director of the largest hospital for epileptic patients in Denmark (700 patients), if any of his patients had an angioma of the face. He informed me of this patient, and a roentgenogram of the skull was made. The picture showed a sinuous shadow in the region of the occipital lobe.

The patient, a woman, was born in 1908. There had been no similar cases in the family; three brothers and sisters were normal. At the age of 3 years, she had an attack of angina, which was followed during the next ten years by seizures associated with unconsciousness, which became gradually more frequent and protracted. Menstruation began at the age of 13, and from that time the fits were accompanied by clonic convulsions, which occurred several times daily, but especially during the night. There was no aura, but involuntary micturition occurred frequently.

Except for the fits, the patient felt well. Mental development was somewhat retarded. In 1921, she was admitted to the Hospital for Epileptics at Dianalund.

Examination.—The nervous system showed nothing abnormal. The pupillary reaction was normal. There was a slight Chvostek sign. The thoracic and abdominal organs were normal. On the right side of the forehead was a somewhat irregular angiomatous nevus, the size of half a hand (fig. 3).

Course.—During her stay at the hospital, the patient was somewhat torpid and was imbecilic; she was periodically depressed and irritable. In 1927, neurologic examination revealed: The pupils were dilated, but reacted well to light and in convergence; the left eye diverged outward; the upper extremities were normal; the knee jerks were normal; the left plantar reflex was not obtained; no Babinski sign was elicited. The spinal fluid contained 20 cells per cubic millimeter, the albumin content was 120 mg. per hundred cubic centimeters, and the globulin content, 10 mg. (somewhat increased); the Pandy reaction was positive; the Wassermann reaction was negative.

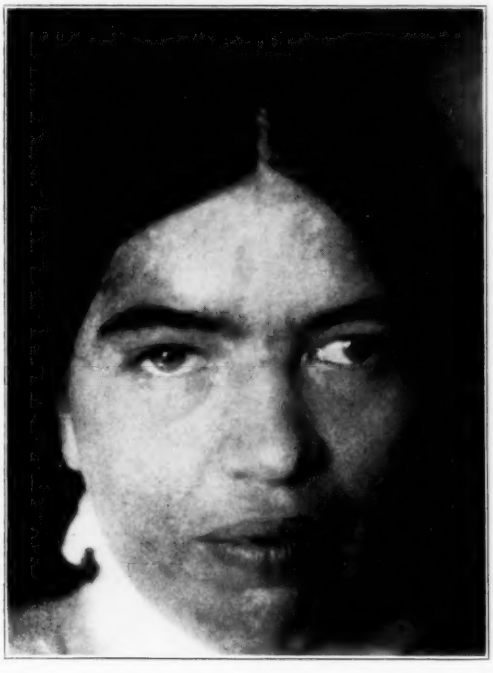

Fig. 3 (case 3).—Angioma of the face.

The basal metabolic rate in 1925, 1927 and 1929 was 85, 88 and 80 per cent, respectively; after treatment with thyroid in 1929, the rate was 106 per cent.

The patient was treated with phenobarbital and sodium borate. In the course of the years she had many minor and some major epileptic attacks. In 1933 she had from ten to fifteen minor attacks each month but only one major attack during the year. She worked at sewing and weaving, at which she was rather clever, but was unsociable and teasing.

Roentgen examination in October 1929 revealed a sinuous shadow corresponding to the lower part of the right occipital lobe, which was prolonged into the temporal lobe. The double contours in the fissures were distinct (fig. 4).

Case 4.—J. H. P. W. was born in 1922, the son of a wholesale merchant. A brother of the paternal grandfather is said to have been epileptic. The father was somewhat degenerated psychopathically. The patient was born normally at term. The weight at birth was about 3,250 Gm. He was breast fed for two weeks only. At the age of 2½ months he had convulsions during three days; the seizures were numerous and characterized by muscular contractions of the face, arms and legs, without cyanosis. On March 30, 1923, he again had numerous slight fits, which were most marked on the right side; they began in the face or arm and lasted for from one-fourth to two hours. The fits continued for a week. Later, fever developed, and he was admitted to the Dronning Louises children's hospital, Copenhagen (Professor Monrad) on April 5, 1923.

Examination.—The skull was somewhat asymmetrical, the left temporal region being somewhat flattened and the right parietal region somewhat prominent. In

Fig. 4 (case 3).—Roentgenogram of the skull.

the left half of the forehead and left eyelid there was a vascular nevus, the size of a hand. In the left parietal region was a similar oblong nevus; on the vertex of the skull was a round nevus, 5 cm. in diameter, and in the upper lip a round nevus, about 1.5 cm. in diameter. The nevi did not protrude above the level of the skin. The fontanel was rather flaccid. There were transitorily a slight Chvostek and a peroneal sign, but the Trousseau sign was absent. The feet were in a slight equinus position and were somewhat edematous. The gaze was inclined to be directed toward the left and upward; it was uncertain whether he could see well. Ophthalmoscopic examination gave normal results. The right pupil was larger than the left; both reacted well to light.

The urine was normal; the temperature was normal; the Wassermann reaction was negative.

Course.—The patient had many attacks of convulsive contraction of the right side of the face and right arm, combined with horizontal nystagmus. Once only

was there observed a slight contraction of the left hand and left foot. The patient seemed not to be affected by the fits.

On Aug. 3, 1928, the patient was admitted to the department for mental diseases of Kommunehospitalet in Copenhagen (Professor Wimmer). The mother said that physical development had been good, but that mentally the boy had been retarded; he had also been very wild and difficult to educate. He had been free from convulsions for half a year after discharge from the Dronning Louises children's hospital. After that, the fits recurred but had more the character of lipothymia—"he fell down like rubber." For the last year he had presented true epileptiform attacks, with unconsciousness, followed by deep somnolence; he would weep on awakening.

Fig. 5 (case 4).—Angioma of the face.

The boy had started to walk at about the age of 2; he had walked with difficulty because of the pes equinus on the right side. He did not speak till the age of 6 years. The teeth appeared at the normal time. He soon became very fat.

Examination at this time revealed a large angioma of the forehead over the left eye, from the median line to 3 cm. lateral to the external angle of the eye and from the eyebrow to the limit of the hair (fig. 5). The angioma was slightly elevated above the level of the skin. The underlying bone was normal to palpation. The ocular movements and pupillary reactions were normal. Ophthalmoscopic examination gave normal results. Speech was very incomplete. The right upper extremity was somewhat shorter than the left; it was flexed at the elbow, and the

forearm was pronated. Muscular power was diminished; the tendon reflexes were increased. The right lower extremity was somewhat aplastic; the foot was plantar flexed. The tendon reflexes were increased. The Babinski sign was positive on both sides. The patient walked with difficulty; gait was spastic, and he walked on his toes. He was very fat. The genital organs were well developed.

During his stay in the hospital, the patient presented marked imbecility. He was, however, able to play with toys. He made small observations, but his behavior was infantile. He kissed the hand of another boy, and wept excessively when his mother left him after her daily visits. He masturbated frequently.

He was discharged from the hospital after nine days.

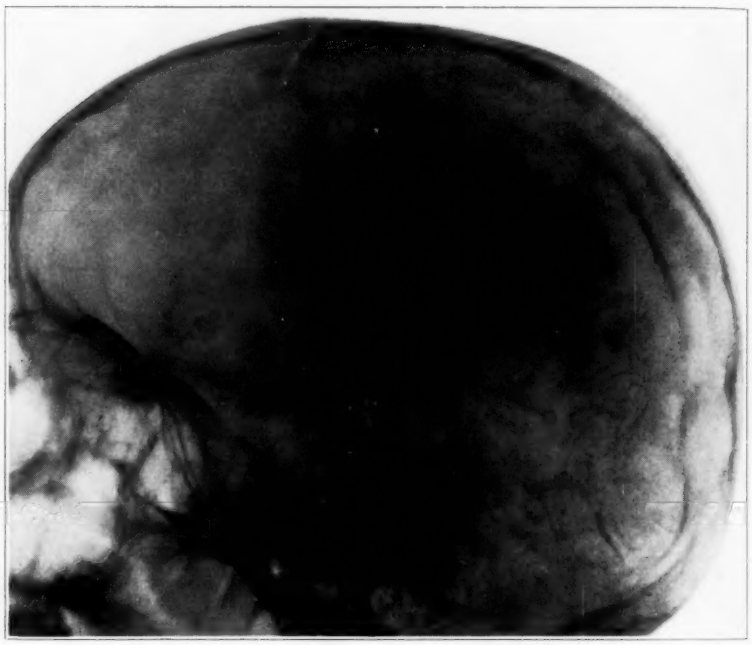

Fig. 6 (case 4).—Roentgenogram of the skull.

A year later he was examined by the resident medical officer of the department. He had been more quiet and spoke somewhat better. The adiposity had increased.

On Nov. 22, 1933, I examined the child, now 11 years of age. He was having recurrent convulsions, generally about four attacks each night. Treatment with phenylethylbarbituric acid had always an unusual effect on him; he became restless and almost maniacal. The gait had been unchanged.

I suspected the presence of an angioma with calcification of the brain, and recommended a roentgen examination, which Dr. Ove Wissing kindly performed. The roentgenogram presented the same characteristic shadow as in the preceding case, a sinuous shadow situated mainly in the region of the occipital lobe (fig. 6).

CASE 5.—The patient was studied by Dr. O. Thranegaard, chief physician at the hospital for epileptic patients at Nyborg, who has kindly submitted his report to me for publication.

History.—H. G., a girl, born on Oct. 26, 1923, who was referred by the Council on Child Welfare, was examined only once. She had terrified the children of her neighborhood; she was aggressive, kicked and scolded, screamed and threw bottles and horse dung about. The parents could not control her. She was said always to have been mentally deficient. From the age of 1 to 6 years, she had been treated with radium for an extensive angioma of the right side of the face and head. Shortly after the initiation of this treatment she had begun to have epileptic fits, sometimes several fits on the same day. It was not clear whether the convulsions were hemilateral, but this did not seem to have been the case. Apart from these symptoms, the girl had been healthy. She has three elder brothers and sisters, all healthy.

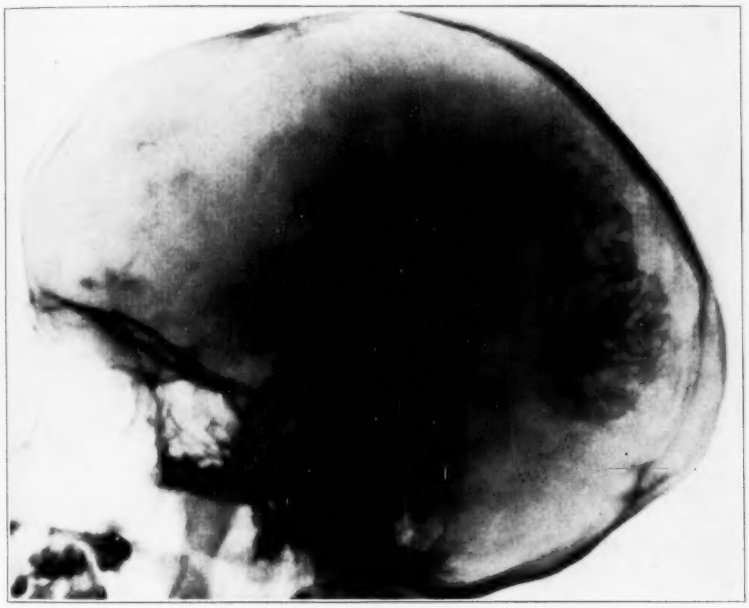

Fig. 7 (case 5).—Roentgenogram of the skull.

Examination.—In the right frontal and temporal region was a very large angioma, which extended to the root of the nose. It was somewhat pale and in spots somewhat cicatrized as the result of treatment. No other angiomas were observed. The right eye did not protrude, and there was no aneurysm in the neck. Ophthalmoscopic examination of the right eye gave normal results; the left eye could not be examined because of a traumatic swelling about the eye. Except for a positive Babinski sign on the left, detailed neurologic examination did not reveal anything abnormal. The patient appeared to be an imbecile.

Roentgen examination showed a characteristic gyriform shadow in the region of the right occipital lobe (fig. 7).

Case 6.—*History.*—J. O., a boy, born in 1912, was treated in the neurologic department of Kommunehospitalet in Copenhagen, in January and February 1927. There was no history of similar cases in the family. The father had been treated in the same department in 1912 for cerebrospinal syphilis. The syphilitic infection, however, occurred nineteen years before the birth of the patient and several years before the father was married. The birth of the patient had been normal. At the age of 9 months he had a convulsion, with fever and vomiting. After that he had no convulsions until 1919. From December 1919 to January 1920, he had influenza with high fever, but without cerebral or pulmonary complications. By the end of February 1920, the patient began to experience mild attacks in which the head turned to the left and he was momentarily unconscious. These attacks, during which he paid no attention to what was said to him, occurred about four times a day. In April 1920, the condition became aggravated; the fits assumed the char-

Fig. 8 (case 6).—Angioma of the right side of the face.

acter of true epileptic fits, with unconsciousness, involuntary micturition and convulsions of the face and limbs. Sometimes the convulsions occurred without loss of consciousness.

During the following years the fits became steadily worse. In 1926 and 1927 he had severe seizures every week. He had absolute amnesia for the severe attacks. Except for the epileptic fits, he felt well. Some years before he had suffered from melancholy and hypochondria, but these symptoms had disappeared. He had attended school, and his behavior had been good. He did poor work in arithmetic. During the last years he had grown rather fat.

Examination.—The patient was adipose (weight about 70 Kg.); the fat was evenly distributed, without special accumulation in the mammary region. The penis appeared somewhat small, but this was due to the adiposity; the testes were of normal volume. The pubic hair corresponded with the age; there was no axillary hair.

The right side of the face was the seat of a large nævus vasculosus, which extended over the right cheek and the right side of the forehead (fig. 8). The teeth were not of the Hutchinson type. Ophthalmoscopic examination gave normal results. The visual acuity was 5/6; the boy was emmetropic. There was some left homonymous hemiamblyopia, but it was impossible to decide the limits exactly. The pupils were large, the left being a little larger than the right; they reacted well to light but relatively less in convergence, probably because of a slight strabismus divergens concomitans with insufficiency of convergence; otherwise the eye movements were normal, and accommodation corresponded with his age. The other cranial nerves presented nothing abnormal; speech was a little hesitating and the soft palate was nearly immobile in intonation.

Fig. 9 (case 6).—Roentgenogram of the skull.

The extremities were normal as regards the joints, tonus, trophic condition, muscular power, coordination, synergy and sensibility. The knee jerks were normal. A Babinski sign was present on both sides. The gait was somewhat heavy. Psychically, the patient seemed somewhat fatuous and infantile.

The systolic blood pressure was 120; the diastolic, 80. The hemoglobin was 91 per cent (Sahli). The basal metabolic rate was normal. The spinal fluid contained 11 cells per cubic millimeter (Fuchs-Rosenthal), no globulin and 50 mg. of albumin per hundred cubic centimeters (normal).

On several examinations the urine showed a feeble reaction for albumin, and microscopic examination revealed some casts.

The roentgenogram (fig. 9) showed an irregular shadow in the region of the right occipital lobe corresponding with the cerebral gyri, with darker lines cor-

responding with the sulci. Some of the darker lines had double contours. The greatest part of the shadow was situated in the lower part of the occipital lobe; a smaller part only was prolonged to the surface of the temporal lobe.

Course.—During his stay in the neurologic department, a nurse observed one fit. The patient lost consciousness for some minutes; clonic contractions occurred, especially in the right arm, and there was involuntary micturition. The temperature was constantly normal; the pulse rate varied between 70 and 100 per minute. The patient was transferred from the neurologic department to that for internal medicine in order that treatment might be administered for the nephritis.

During the following two years the condition did not change greatly. Three minor fits occurred as a rule each day and two major fits each night. In the minor fits the patient fell, turned his eyes to the left, moaned and had tonic con-

Fig. 10 (case 6).—Photograph of the brain, showing a sclerotic right occipital lobe (*x*).

tractions of the arm. During the major nocturnal attacks he was completely unconscious and presented universal clonic contractions and often involuntary micturition.

In 1929, the diurnal and nocturnal attacks became more frequent; the dose of phenobarbital was increased, and the attacks became more rare. The patient became more and more slow, dull, irritable and choleric.

In spite of dietetic treatment the urine continued to give an albumin reaction. After each fit, the patient complained of headache; except for this, however, he did not have headaches, giddiness or vomiting. Sometimes he had transitory attacks of amblyopia.

In November 1931 the condition became worse. The patient had an abscess in the right axilla, and began to experience severe nausea, vomiting, anorexia, headache and pains. He became more feeble and dull, and did not speak. There was, however, no paralysis.

On Nov. 24, 1931, the patient was again admitted to the neurologic department of Kommunehospitalet. Examination revealed nothing new. Ophthalmoscopic examination showed the disks to be somewhat paler than normal. Some myoclonic contractions were noted. The urine contained significant quantities of albumin, and the blood urea was increased to more than 250 mg. per hundred cubic millimeters of blood. The face was edematous. Several epileptic attacks occurred, and on Dec. 1, 1931, the patient died.

Autopsy.—There were: chronic hemorrhagic nephritis, chronic hyperplasia of the spleen, fatty degeneration of the liver, left-sided fibrinous pleurisy and infarcts of the left lung.

The Brain: The shape and size, as a whole, were normal; the right occipital lobe alone was atrophied, with narrow gyri, and furthermore this lobe was firm (fig. 10). The dura and arachnoid were normal. The pia showed no true

Fig. 11 (case 6).—Roentgenogram of the brain.

angiomatosis; over the atrophied occipital lobe and some parts of the right hemisphere, especially the temporal lobe, it was darker than normal and seemed more richly vascularized. This dark color seemed to be due to an abundant capillary vascularization. The roentgenogram presented shadows corresponding with those that had been found in the roentgenogram of the head in vivo (figs. 11 and 12). Sections of the occipital lobe revealed that the cortex and the corona radiata were atrophied and darker than normal. Fine granules of the lime salts were seen and felt; it seemed even macroscopically, as if the calcifications were situated in the outer layer of the cortex, and not in the pia mater. This observation was confirmed by microscopic examination.

The brain was fixed in toto in formaldehyde and formaldehyde was injected into the carotid arteries. Specimens were stained with iron hematoxylin and eosin and with hematoxylin and picrofuchsin and by the myelin sheath method of Weigert, Kulschitsky and Wolters, the glia-staining method of Holser and the axis-cylinder-staining method of Bielschowsky.

Fig. 12 (case 6).—Roentgenogram of the brain.

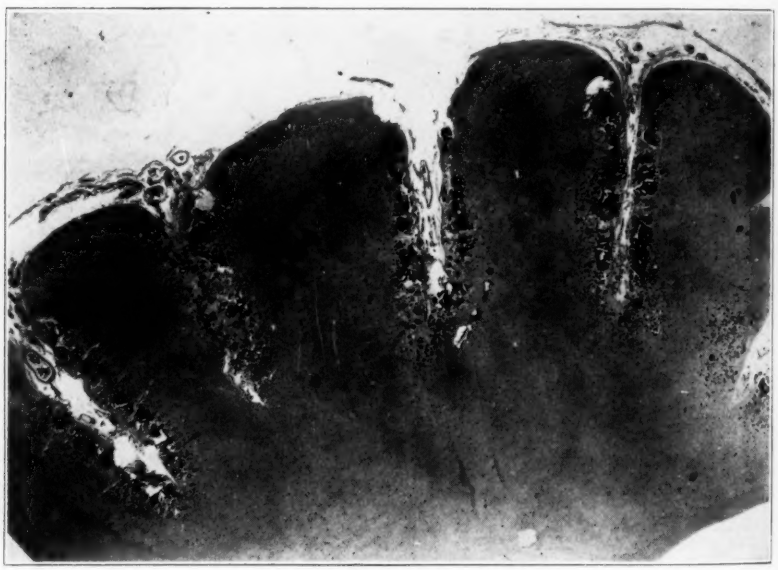

Fig. 13 (case 6).—Section from the sclerotic right occipital lobe, showing calcifications of the outer layer.

Microscopic study showed: In the right occipital lobe, presenting the calcifications, the arachnoid was somewhat thickened, and rich in cells. The pia mater did not contain any calcification, either in the connective tissue or in the vessels. In some areas, but not everywhere, the pia mater was richly vascularized and somewhat angiomatous. I have compared the preparations with some from true angiomas in the pia (kindly lent to me by Professor Kalischer). In these the pia was filled with numerous thick, tortuous, compact masses of vessels. In my preparations the vascularization was not so compact.

All the calcifications were situated in the cortex cerebri (fig. 13). This presented a strange appearance, being scattered all over with granules of lime salts, which varied in size and shape. The smallest granules were globular or slightly angulate; the larger ones seemed to be conglomerated from smaller ones, thereby

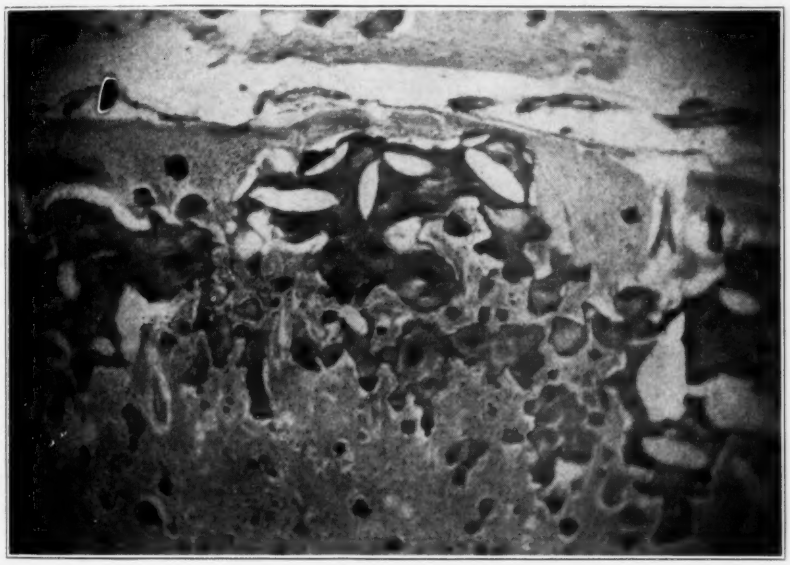

Fig. 14 (case 6).—Section from the calcified outer layer of the right occipital lobe.

presenting a mulberry-like or coralline aspect (fig. 14). The largest granules were visible macroscopically, being some decimillimeters in diameter; from this they decreased in size until they became scarcely visible. The somewhat larger granules were stratified.

The granules were situated for the most part in the second and third layers of the cortex, but some were present also in the molecular layer. In the deeper layers, as well as in the medullary substance, only a few scattered granules were visible. The granules had no relations with the vessels; on the whole, the vessels appeared normal and showed no calcification.

Corresponding with the calcified areas, the number of nerve cells was considerably reduced. In the second layer and outer part of the third layer the nerve cells were entirely absent; the fourth, fifth and sixth layers presented rather well preserved nerve cells, but they were disarranged.

The neuroglia was enormously increased. The granules of lime salts, especially, were embedded in a dense layer of fibrillar neuroglia.

The myelin substance appeared generally normal, containing few granules of lime salts and presenting only here and there some slight decolonization. The myelinated fibers from the fifth and sixth layers presentead the same appearance.

In the right temporal lobe there was a more marked angiomatous formation in the pia mater (fig. 15). On the other hand, the gyri were not so aplastic as were the right occipital gyri. They presented calcifications, but these were much smaller and fewer than those in the occipital lobe. Furthermore, the increase of the neuroglial tissue was not so striking.

Specimens from the right central and the left occipital lobe were normal.

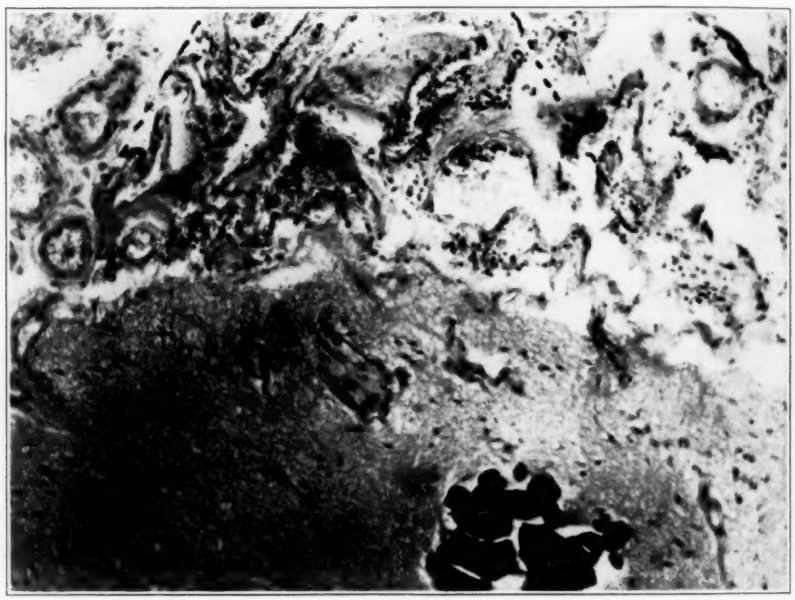

Fig. 15 (case 6).—Section from the right temporal lobe, showing an isolated calcification and angiomatosis of the pia mater.

SUMMARY AND CONCLUSIONS

The cases reported present a characteristic clinical picture, nearly all showing a combination of epileptic fits and angiomas of the face. To these, the most prominent symptoms, may be added as frequent symptoms: mental debility and a slight spastic hemiplegia on the side opposite to the angioma of the face. Furthermore, the patients are sometimes rather fat. If roentgen examination is made, a characteristic shadow appears inside the skull: a distinct sinuous shadow, presenting exactly the shape of the surface of the brain, showing both gyri and sulci, often with double contours. Generally, this shadow is localized in the occipital lobe.

Histologic examination of the brain shows that the shadow is due to calcification of the outer layers of the cortex, not of the pia mater. This presents in some parts an abundant vascularization, but not a true angioma. The calcification consists of numerous small, mostly microscopic, granules of lime salts, localized in the second and third layers of the cortex. In these layers the nerve tissue is in great part destroyed and replaced by fibrillar neuroglia. In the case described, the occipital lobe, in which the processes were localized, had shrunk and was sclerotic. Otherwise the brain seemed fairly normal; only in places were the same modifications seen to a very slight degree.

The changes in the brain probably are not secondary to angiomas of the pia mater. The syndrome must be related to a more generalized malformation of the organism (as in tuberous sclerosis), consisting in the formation of angiomas of the face, slight angiomatous modifications of the pia mater, aplasia of the occipital lobe of the brain with (probably secondary) sclerosis and calcification of the aplastic part. The malformations probably originate in fetal life.

The anatomopathologic observations suggest certain directions for therapy. The calcifications are related not to a tumor but to a congenital defect and malformation. Hence, operation is not indicated. Roentgen treatment may perhaps be administered "consolaminis causa." No striking results are to be expected from treatment, however, since regeneration of aplastic brain tissue is not possible. Symptomatic treatment for the epileptic fits and mental hygienic treatment for the mental defects must be considered as the only therapy.

If this disease, forming an entity, should deserve a name, it might well be: Parkes Weber-Dimitri's disease.

CHANGES IN CUTANEOUS LOCALIZATION IN A PEDICLE FLAP

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AND
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Kredel and Evans [1] recently reported results of a study of the return of sensation to various types of skin grafts, especially pedicle flaps. In all their cases, however, the original nerve supply to the transplanted skin was severed completely, and consequently there was no instance of "reference" of sensation to its former locus. We have not, in fact, seen a report of a case in which enough of the original nerve supply remained and in which at the same time an adequate shift of locus occurred to produce "false" localizations of this sort.[2] In view of the comparative crudeness of localization in all except the very motile parts of the body, the rarity of such a phenomenon is not surprising. The present report is based on the study of a case in which both requirements essential to anomalous localization in such transferred flaps of skin were satisfied, and in which, consequently, sensations were at first referred to the original site of the pedicle flap.

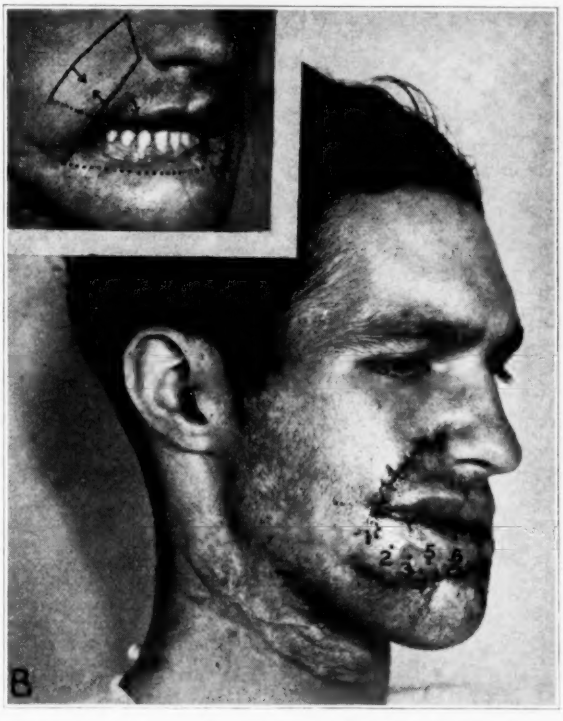
From the Department of Surgery, School of Medicine and of Psychology, College of Arts and Science, Vanderbilt University.

1. Kredel, F. E., and Evans, J. P.: Recovery of Sensation in Denervated Pedicle and Free Skin Grafts, Arch. Neurol. & Psychiat. **29**: 1203 (June) 1933.

2. We failed to see the recent article by Purdy (Tactual Space Perception in Translocated Tissue, J. Gen. Psychol. **10**:227, 1934) until after the present report was written. Purdy described a case in which reorientation to a segment of transferred skin failed to develop. His patient suffered an injury to the middle finger of the left hand, necessitating the removal of the whole terminal phalanx except a large part of the skin of the volar surface. This skin was folded back over the exposed joint and stitched to the skin of the dorsal side of the second phalanx. Stimuli applied to the displaced skin have been invariably localized in illusory fashion for ten years, in contrast to the case reported here, in which reorientation developed within a year. This discrepancy is probably due to the fact that in Purdy's case the transferred skin was not shifted into an area possessing perceptual-motor habits decidedly different from those required for adjustment to the original locus, whereas in our case the cutaneous field surrounding the transferred flap possessed a "local sign" quite different in character from that of skin bounding the flap in its former site. Furthermore, the importance of accurate adjustment to such an area as the lower lip seems to require the development of relevant perceptual-motor habits.

REPORT OF A CASE

A white man, aged 19, was thrown from a motorcycle against a barbed-wire fence on Jan. 28, 1933. The right side of the face, lips, tongue and neck were severely lacerated. The entire right four fifths of the lower lip was destroyed, exposing the lower teeth and preventing full closure of the mouth. The patient could neither speak clearly nor prevent saliva from running down the surface of his chin. At the time he entered the Vanderbilt Hospital the wounds had all healed. The main problem was that of building a new lower lip to replace the one which had been destroyed. The operation consisted in a complete one-stage



A, loss of substance of lower lip and outline of pedicle flap from cheek used to replace it; *B*, flap forming new lip sutured in place, wound of cheek closed. The numbers indicate the points used in the studies on contact stimulation.

reconstruction of the deficient portion of the lower lip, including the mucous membrane lining it.

Reference to the small insert in the illustration will enable one to follow the procedure used in this operation. A finger-shaped or U-shaped flap of skin, subcutaneous fat and muscle, represented by the solid lines, was raised by sharp dissection from the right nasolabial region between the corner of the mouth and the right nostril. This flap was shifted downward 45 degrees in the direction indicated by the lowest arrow, to the position shown by the dotted line. The flap was left attached through its base or pedicle to the right cheek, while its free end

was sewed to the freshened edge of the left side of the lower lip, and its lower edge to the freshened upper edge of chin tissue. Its upper edge and inner surface were covered by a flap of mucous membrane swung around from the inner surface of the right cheek. The edges of the wound on the right cheek, left by the removal of the flap, were approximated by undermining and suturing. This step is indicated by the two upper arrows. The entire operation was performed with the aid of procaine hydrochloride block and infiltration analgesia.

The larger photograph shows the completed operation. The lower finger-shaped line of sutures represents the flap shifted from above but left attached to the cheek. The upper line represents the closure of the area from which the flap was removed. The operative wounds healed in twelve days.

The pedicle flap was originally innervated by nerves from two branches of the fifth cranial nerve: the buccinator nerve, derived from the mandibular branch, and the superior labial branch of the infra-orbital division of the maxillary nerve. The incision severed all the nerve supply to the area derived from the latter source, leaving the fibers from the mandibular intact. A few days after the operation the patient reported that sensations arising from sensitive parts of the new lower lip were localized on the upper lip or on the cheek. The decided character of the "reference" suggested the desirability of a series of localization tests, for the purpose of following the development of reorientation to the newly formed lip. Accordingly, the six points shown in the illustration were selected for study, the configuration of scars on the flap making it possible to locate these points easily.

Judging by the sensitivity of different parts of the flap immediately after the operation, the latter was well supplied with nerve fibers for more than half the distance from the base or pedicle toward the free end. Thresholds of contact stimulation for points 1, 3 and 5 were 0.5, 0.57 and 7.7 Gm., respectively, as measured by a specially constructed limen gage. Beginning at point 3 sensitivity declined rapidly, apparently indicating the limits of the receptive field of the buccinator nerve fibers in the flap. Points 4 and 6 were practically insensitive to pressures up to 10 Gm., which was the maximum used.

METHOD OF PROCEDURE

In the localization tests the subject was stimulated with a constant pressure of 15 Gm. One tip of the Ebbinghaus double esthesiometer was used as the stimulating point. The tip is of ebony, rounded at the point so as to avoid pain from pricking the skin. The subject lay blindfolded on a table. After the stimulus was applied and removed the subject was asked to describe as accurately as possible the location of the pressure sensation. Certain categories of loci were agreed on, the principal ones being: "upper lip," "upper cheek," "corner of mouth" and "lower lip." The subject was urged to qualify or amplify his descriptions by way of indicating more precisely the apparent location of the stimulus within these four general areas. Under the circumstances it seemed desirable to use this method of response rather than the more generally used method of Weber, in which the blindfolded subject tries to touch the point stimulated with a stylus. Although the latter method yields quantitative values, the configuration of the areas would probably have made them relatively meaningless in the present case. Furthermore, this method involves both kinesthesis from moving the arm and hand, and confusing contact sensations when the stylus carried in the subject's hand touches the skin of the general area being stimulated. Both these factors complicate the psychologic process involved in the perception of the locus of stimulation, especially

for an unpracticed subject. It was thought that the use of the four categories, which were derived from the subject's descriptions in preliminary tests, would be adequate to display changes in localization in the pedicle flap. Eight trials, on the average, were made for each point at each of the five examinations, with the exception of point 5, which was not tested at the fourth examination owing to a small infection at that spot. Points 4 and 6 were almost totally anesthetic at the first examination, with respect both to cutaneous and to deep pressure sensibility.

RESULTS

The figures in the accompanying table summarize the essential results of the study. Data from two groups of points are: 1. Points 1, 2 and 3 retained practically normal contact sensitivity. 2. Points 4 and 6 were anesthetic following the operation. Results for the individual points within each group all manifested the same trend, although the specific figures naturally varied from the averages shown. The figures

Percentages of Localization for the Two Groups of Points Within Each of the Four Areas Used to Designate the Apparent Locus of Stimulation

Days After Operation	Points 1, 2 and 3*				No Response	Points 4 and 6*			
	UC	UL	CM	LL		UC	UL	CM	LL
11	39	44	17	0	100	0	0	0	0
41	7	41	45	7	28	0	0	11	61
143	4	0	67	29	0	0	0	31	69
265	0	0	15	85	0	0	0	0	100
325	0	0	0	100	0	0	0	0	100

* These response categories were "upper cheek" (UC), "upper lip" (UL), "corner of mouth" (CM) and "lower lip" (LL). The points are numbered in the illustration.

for point 5 were omitted, since no test was made there on the fourth examination. The values in the table are percentages of localizations reported by the subject in terms of the four general areas mentioned. In the case of the first group of points the figures show clearly that at the first examination none of the stimuli on the sensitive part of the pedicle flap were correctly localized. The contact sensations were referred to the upper lip, to the adjacent upper part of the cheek and to the corner of the mouth (or "out from the corner"). In the four subsequent trials, over a period of almost eleven months, there was a progressive shift of "local sign" to the lower lip. The process of transition was apparently gradual, involving considerable confusion for the subject. The tendency to localize most of the stimuli at the corner of the mouth at the third examination (one hundred and forty-third day from the operation) may perhaps be interpreted as a sort of "resolution" of the conflict between the old and the newly developing orientation habits. This confusion may have been conditioned by the ingress of new fibers from the chin adjacent to the lower border of the flap, although the fact that at this stage localization was at the corner of the mouth, rather

than on the lower lip or chin, and the further fact that the sensitivity of these points remained normal throughout the experiment, suggest that reorientation involving stimulation of the intact fibers entering the base of the flap is the more probable explanation.[3] By the fourth test, on the two hundred and sixty-fifth day, the subject localized 85 per cent of the stimulations on the lower lip, showing the growing strength of the new habit. At the last test, on the three hundred and twenty-fifth day, all the stimuli were correctly localized on the lower lip. At this examination, when it appeared that the subject was localizing the sensations on the lower lip without difficulty, Weber's method of pointing to the spot stimulated was used. The subject's average error, for ten trials at each of these three points, was 6 mm., in comparison with an error of 3 mm. secured for analogous points on a normal subject.

The results for points 4 and 6, which were anesthetic following the transfer of the flap to the lower lip, present an interesting contrast with those just given for points 1, 2 and 3. At the first examination the subject failed to respond at all to the 15 Gm. stimulus at point 4 and felt only a single diffuse, unlocalized pressure at point 6. Apparently, both cutaneous and deep pressure fibers were absent to such an extent that even crude localization was impossible. At the second test, on the forty-first day, nerve fibers apparently had entered this part of the flap, since 72 per cent of the stimuli were felt and localized. But most of the sensations were localized on the "lower lip," whereas at the same sitting most of the localizations for the other group of spots were on the upper part of the cheek, upper lip or at the corner of the mouth. Evidently, most of these "new" nerve fibers innervating spots 4 and 6 came from the mental branch of the mandibular nerve, which supplies fibers to the cheek and to the residual part of the lower lip to which the flap was attached. Consequently there was no "reference" of sensation to the upper lip or cheek. The localization of a small percentage of sensations at the corner of the mouth, at this test and in the succeeding one, was probably due to the "dulness" of the contact sensations at this stage of

3. In order to check the possibility that the shift in localization may have been due to the ingress of nerve fibers from the chin bordering the flap, the following experiment was performed: The skin along the line of the flap was infiltrated with a 1 per cent solution of procaine hydrochloride, and the pain sensitivity of the flap tested. The blocking of nerve impulses from the adjacent part of the chin did not affect the sensitivity to pain in the portion of the flap which retained sensitivity following the operation, although the sensitivity of the tip was reduced. Next, the skin across the pedicle was infiltrated with procaine hydrochloride and the sensitivity to pain was again tested. The latter was abolished from the line of infiltration out almost to point 5; from this point to the tip the sensitivity to pain was almost normal. Both sets of results fully confirm our interpretation of the neural basis of the shift in localization.

nerve growth and to the consequent difficulty of definite localization. The subject reported such "dulness" and confusion in several instances.

It is interesting to compare the two groups of points with respect to the time required for the growth of accurate perception of sensation-locus. The figures in the table show clearly that more time was required for the development of reorientation to points 1, 2 and 3 than for the ingress of new nerve fibers into the free end of the flap from adjacent tissues.

COMMENT

The results of this study support the view that "local sign" depends mainly on habit formation, in which muscular adjustments to various body areas and the resulting kinesthesis play the primary rôle. Our subject had to build up new perceptual motor habits with reference to the part of the pedicle flap which retained its original nerve supply. In this process, kinesthesis resulting from manual exploration and sensations arising from contact of the lower with the upper lip were no doubt the essential factors. Considerable confusion resulted from conflict of the old with the new orientation habits. A consequence of this conflict apparently was a tendency to localize the majority of the stimuli for a time in an area intermediate between the old and the new locations of the pedicle flap. The confusion which the subject often manifested and reported appears to justify the interpretation of this "intermediate stage" as due to a sort of compromise or resolution of the conflicting orientation tendencies. On the other hand, for the part of the flap which retained little or none of the original innervation there was no tendency to refer sensations to the former locus of the transferred flap.

The results of the present study are analogous to those secured by Stratton [4] in his well known experiment with lenses which reversed the usual relations of visual images on the retina. While covering one eye, Stratton placed before the other an optical instrument which produced upright images of objects on the retina. This instrument was worn continuously for a week, except at night when both eyes were covered. Great confusion naturally occurred at first, but by the end of the week all motor adjustments were properly coordinated with the new visual relations. When the instrument was removed a new period of false movements and inverted perceptions supervened before the old habits were reestablished. The shorter period of reorientation in Stratton's experiment, in comparison with the present case, was no doubt due to the greater necessity and practice of the visual motor adjustments.

4. Stratton, G. M.: Vision Without Inversion of the Retinal Image, Psychol. Rev. **4**: 341 and 463, 1897.

Bodily "local signs" like retinal "local signs" are, as Peterson [5] has said, habitual "orientation tendencies" conditioned by the motor adjustments of a subject to the spatial relations involved.

SUMMARY

1. A case is presented in which a pedicle flap from the right nasolabial region was substituted for the right four fifths of the lower lip, the latter having been destroyed in an accident.

2. The dissection of the flap resulted in denervation of the outer fourth (roughly) of its free end, but the original nerve supply entering from the pedicle was left intact and sensations from this sensitive portion were referred mainly to the upper lip and cheek.

3. Results of localization tests carried out on the sensitive part of the flap revealed a progressive shift of "local sign" from the upper lip and cheek to the lower lip. The development of reorientation required several months. A transitional stage was observed, involving considerable confusion and a tendency to localize sensations in an area intermediate between the old and the new locations of the flap. This tendency was interpreted as a resolution of the conflict arising between the old and the new orientation habits.

4. No "reference" was observed on return of sensitivity to the denervated portion of the pedicled flap. The ability to localize sensations within this area began to return within six weeks and was apparently coincident with the ingress of nerve fibers from the edges of the chin and lip to which the flap was attached.

5. These results clearly indicate that "local sign" depends mainly on habit formation, involving the association of cutaneous impulses with the kinesthetic impulses.

5. Peterson, Joseph: Local Signs as Orientation Tendencies, Psychol. Rev. **33**: 219, 1926.

HISTOPATHOLOGY OF THE EXTERNAL GENICULATE BODY

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The external geniculate body has been studied extensively in numerous investigations of the central visual system, but such research has been concerned largely with the anatomy of the fibers and the representation of the retina in the central nervous system. A consideration of the cellular pathology of this structure, particularly with regard to the effect produced by inflammatory, toxic or degenerative processes, was not found in a review of the literature. Since the external geniculate body is directly associated with the function of vision, the study of its cellular pathology reported here has revealed changes of both academic and clinical interest.

MATERIAL AND METHODS OF STUDY

The material from one hundred and forty-one cases of a wide variety of infectious, toxic and degenerative conditions was examined. In most of the cases the external geniculate body was sectioned from one side and stained by the Nissl, fat, myelin sheath, neurofibril and glia fiber methods, depending on the needs in each case, the Nissl stain being the most important. For normal anatomic and histologic orientation, serial sections through the ganglion were made in three cases and stained by the Nissl method. The observations in eight normal cases and seven cases of schizophrenia were used as examples of normal histologic conditions to compare with the pathologic material.

ANATOMY AND HISTOLOGY

Anatomic details of the structure of the geniculate body can be found in the studies of Minkowski,[1] Malone,[2] Brouwer,[3] Putnam[4] and,

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From the Deutsche Forschungsanstalt für Psychiatrie, Munich, Germany.

1. Minkowski, M.: Ueber den Verlauf, die Endigung und die zentrale Repräsentation von gekreuzten und ungekreuzten Sehnnervenfasern bei einigen Säugetieren und bei Menschen, Schweiz. Arch. f. Neurol. u. Psychiat. **6**:201, 1920.

2. Malone, E.: Ueber die Kerne des menschlichen Diencephalon, Neurol. Centralbl. **29**:290, 1910.

3. Brouwer, B.: Experimentelle anatomische Untersuchungen über die Projektion der Retina auf die primären Opticuszentren, Schweiz. Arch. f. Neurol. u. Psychiat. **13**:118, 1923.

4. Putnam, T.: Studies on the Central Visual System: I. The Anatomic Projection of the Retinal Quadrants on the Striate Cortex of the Rabbit, Arch. Neurol. & Psychiat. **16**:1 (July) 1926; II. A Comparative Study of the Form of the Geniculostriate Visual System of Mammals, ibid. **16**:285 (Sept.) 1926; III. The General Relationships Between the External Geniculate Body, Optic Radiations and Visual Cortex in Man, ibid. **16**:566 (Nov.) 1926; IV. The Details of the Organization of the Geniculostriate System in Man, ibid. **16**:683 (Dec.) 1926.

more recently, Balado and Franke.[5] This small ovoid mass of gray matter is located in the posterior course of the optic tracts, and can be seen as a swelling at the point at which the optic tracts curve around the lateral aspect of the cerebral peduncles. It receives the major portion of the fibers from the optic tract, and sends fibers which form the optic radiations to the striate cortex. The cellular portion averages 8 mm. in length in the pyroxylin (celloidin) serial sections, with a volume estimated by Putnam to be about 0.11 cc. It is bounded above and medially by the posterolateral thalamus, and below and laterally by the cornu ammonis. The medial geniculate body and the cerebral peduncles lie medially and inferiorly. The shape of the ganglion, as well as its relationship to these structures, varies according to the portion sectioned.

In transverse serial sections beginning in the optic tract the cells of the external geniculate body are first seen as a small cluster of medium-sized ganglion cells densely packed together and surrounded by fibers of the optic tracts. As sections are taken posteriorly this mass of cells becomes larger and the proportion of optic fibers relatively less. In the ventral region the cells become larger and soon separate from those above to form a ventral layer of large cells. In the anterior third of the ganglion the optic fibers begin to pass around the mass of cells, dividing them into more or less sagittal bundles. In the middle third the typical arrangement of the cells into concentric rows separated by white fibers takes place, giving an easily identified and characteristic architectonic structure. Here a ventral hilar groove makes the cell layers assume the shape of an inverted V over the hilus. Typically, one finds in this region two ventral rows of large cells, above which lie four rows of smaller cells. The typical arrangement described by Minkowski in apes agrees in general with the description given by Balado and Franke in man. Minkowski recognized three types of cells. The large cells, found chiefly in the ventral rows, are triangular, oval or spindle-shaped, with large nuclei and prominent processes. They contain much tigroid substance arranged concentrically around the nucleus. In the other layers most of the cells are medium-sized or small. These cells are more oval than the large cells, with less prominent processes. They have round, clear nuclei, and are less rich in Nissl granules. All the cells are lipophilic. Some of the layers contain a mixture of the three types of cells. In the posterior third of the

5. Balado, M., and Franke, E.: On the Structure of the Lateral Geniculate Body in Man, Rec. Soc. argent. biol. **5**:419, 1929; abstr., Zentralbl. f. d. ges. Neurol. u. Psychiat. **56**:247, 1930. The Corpus Geniculatum Externum of Man, Bol. Inst. clin. quir., Univ. Buenos Aires **7**:5, 1931; abstr., Zentralbl. f. d. ges. Neurol. u. Psychiat. **62**:710, 1932. Histologic Structure of the Layers of the Corpus Geniculatum Externum of Man, Bol. Inst. clin. quir., Univ. Buenos Aires **7**:21, 1931; abstr., Zentralbl. f. d. ges. Neurol. u. Psychiat. **63**:418, 1932.

ganglion the hilus, which divides the rows of cells into a medial and a lateral limb, gradually disappears, and the cells of the lateral limb extend farther posteriorly, but maintain their layered arrangement to end abruptly in the pulvinar of the thalamus.

The external geniculate body receives its blood supply from small vessels that enter along the hilus. Duret,[6] Beevor,[7] and Foix and Hillemand [8] found these small vessels to be branches of the posterior cerebral artery. The lateral portion of the ganglion is supplied by a branch of the anterior choroid artery, according to Dejerine,[9] Foix [10] and Abbie.[11] A branch of the posterior cerebral artery which arises at the place at which the posterior cerebral artery passes under the medial geniculate body was called by Foix and Hillemand the pediculus posterolateralis externus. This small vessel sends from four to six branches into the hilus, and supplies the medial portion of the external geniculate body and the lateral portion of the medial geniculate body. It penetrates into the posterolateral thalamus. This may be strengthened by a branch from the posterior choroid artery. Pellicer [12] found the capillary net variable, but in general the cellular layers were more vascular than the white matter.

GENERAL HISTOPATHOLOGY

The cells of the external geniculate body were not sensitive to changes post mortem when compared with other cell groups, as, for example, the cells of the dentate nucleus of the cerebellum, which according to Scherer,[13] show rapid changes post mortem.

As would be expected, the most severe damage to the ganglion cells of the lateral geniculate body was found in the most acute disease processes. Thus, "severe cell disease" (used in the sense described by Spielmeyer [14]) was found in pertussis, meningitis, uremia and status epilepticus when the disease was of great intensity. The changes in the ganglion cells were accompanied by progressive and regressive reac-

6. Duret: Arch. de physiol. **6**:71, 82 and 85, 1874.

7. Beevor, C. E.: The Cerebral Arterial Supply, Brain **30**:403, 1907.

8. Foix, C., and Hillemand, P.: Les artères de l'axe encéphalique jusqu'au diencéphale inclusivement, Rév. neurol. **2**:705, 1925.

9. Dejerine: Anatomie des centres nerveux, Paris, J. Rueff, 1901, p. 165.

10. Foix, C., cited by Misch, W.: Die cerebralen Gefässverschlüsse und ihre klinischen Syndrome, Zentralbl. f. d. ges. Neurol. u. Psychiat. **53**:673, 1929.

11. Abbie, A. A.: The Clinical Significance of the Anterior Choroidal Artery, Brain **56**:233, 1933.

12. Pellicer, T. R.: The Structure of the External Geniculate Body, Trav. lab. biol., Madrid **25**:319, 1928; abstr., Zentralbl. f. d. ges. Neurol. u. Psychiat. **52**:10, 1929.

13. Scherer, H. J.: Beiträge zur pathologischen Anatomie des Kleinhirns, Ztschr. f. d. ges. Neurol. u. Psychiat. **139**:337, 1932.

14. Spielmeyer, W.: Histopathologie des Nervensystems, Berlin, Julius Springer, 1922, p. 67.

tions of the neuroglia, as is usually the case. The cells of the olivary body, which are strongly lipophilic like those of the lateral geniculate body, also show severe cellular disease in the most acute processes, according to Braunmühl.[15]

The tendency of the cells of the external geniculate body to undergo pigment atrophy must be strongly emphasized. This was by far the most common change accompanying a wide variety of disease processes in the subacute and chronic stages. The pigment atrophy was associated with the so-called fatty sclerosis of some of the cells and a variable degree of progressive and regressive changes of the fixed glial elements, especially the progressive protoplasmic form of the macroglia cells. The small blood vessels also showed an excessive accumulation of pigment in the endothelial and adventitial cells. This accumulation of lipoid pigment in the cells is apparently their method of reaction to a noxious agent of subacute intensity, and demonstrates the nonspecificity of the reaction of ganglion cells to subacute intoxication first emphasized by Nissl.[16] It may be interpreted as an acceleration of normal cellular involution, as all lipophilic cells normally accumulate more and more pigment with advancing age. It was therefore necessary to consider the age of the patient as well as the disease process present before concluding whether the accumulation of pigment was abnormal.

In some of the sclerotic diseases of the nervous system, as well as in the more chronic infectious and toxic conditions, the degree of pigment atrophy appeared to be out of all proportion to that found normally at the same age (the normal values were determined in a previous study [17]). This was often true in such conditions as the chronic stage of epidemic encephalitis, dementia paralytica, cerebral syphilis, carbon monoxide poisoning, anemia and diabetes. In the group of senile and arteriosclerotic conditions, the pigment atrophy in some of the cases was excessive in relation to the age of the patients. In other chronic conditions like multiple sclerosis, Pick's disease and paralysis agitans, the degree of pigment atrophy corresponded more to that seen in senile persons, although the patients were young.

SPECIAL HISTOPATHOLOGIC CONSIDERATIONS

In some conditions the pathologic changes in the external geniculate body were of sufficient interest to deserve special consideration.

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15. von Braunmühl, A.: Ueber Ganglienzellveränderungen und gliöse Reaktionen in der Olive, Ztschr. f. d. ges. Neurol. u. Psychiat. **126**:621, 1930.

16. Nissl: Ueber die Veränderungen der Nervenzellen nach experimentell erzeugter Vergiftung, Neurol. Centralbl. **14**:947, 1896.

17. Steegmann, A. T.: A Peculiar Condition in the Cells of the External Geniculate Body Resembling Amaurotic Idiocy, Arch. Neurol. & Psychiat. **31**:701 (April) 1934.

Encephalitis.—In various types of acute encephalitis lesions were usually present in the lateral geniculate body, so that one can conclude that the gray matter in this region is rarely spared in the acute stage of the disease.

Figure 1 shows the encephalitic process in a case of epidemic encephalitis. The inflammatory process destroyed the medial portion of the large cell layers in the posterior portion of the ganglion, and the large cells had almost completely disappeared. An occasional ganglion cell showed a peculiar homogeneous change, with a displaced kidney-shaped nucleus.

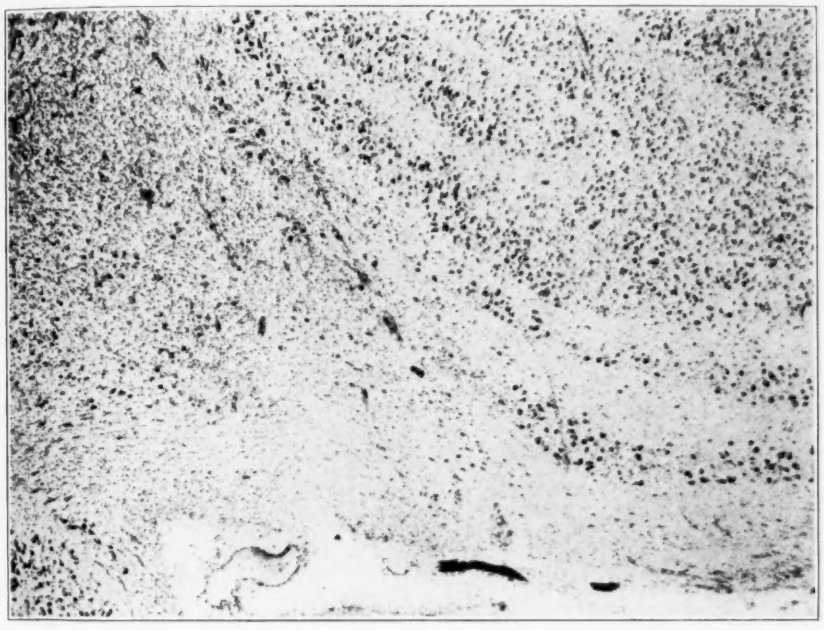

Fig. 1.—Inflammatory process in the lateral geniculate body in subacute epidemic encephalitis. Part of the ventral large cell layers are destroyed. The intensive glia reaction is visible. Nissl stain.

In addition to the perivascular round cell infiltration, the increase of neuroglia was evident. This consisted of progressive macroglia (astrocytes), rod cells and lipoid-filled regressive glia cells with shrunken pyknotic nuclei. The ganglion cells apart from the focal inflammatory area had undergone chromatolysis and swelling, a change described by von Economo [18] in one of his early publications.

According to Spatz,[19] the region of the pulvinar and medial geniculate body is often affected in the acute stage of epidemic encephalitis, but shows few defects

18. von Economo, C.: Die Encephalitis lethargica, Vienna, Franz Deuticke, 1918.

19. Spatz, H.: Encephalitis, in Bumke, O.: Handbuch der Geisteskrankheiten, Berlin, Julius Springer, 1930, vol. 11, p. 251.

in the end-stage. This was found to be true also in the lateral geniculate body in the acute and chronic cases studied.

In postvaccinal encephalitis, localized inflammatory foci were found in both the cellular and the acellular portion of the external geniculate body in all the cases in which sections were made. These foci were similar to those in the gray and white matter throughout the nervous system and were, as a rule, perivenous in distribution. Their histology is well known, and needs no special description.

The lateral geniculate body may be the site of metastatic foci of encephalitis, shown in figure 2. The focus occurred in a case of sepsis following tonsillitis. It is similar to the lesions described by Diamond [20] in cases of septicemia and pyemia.

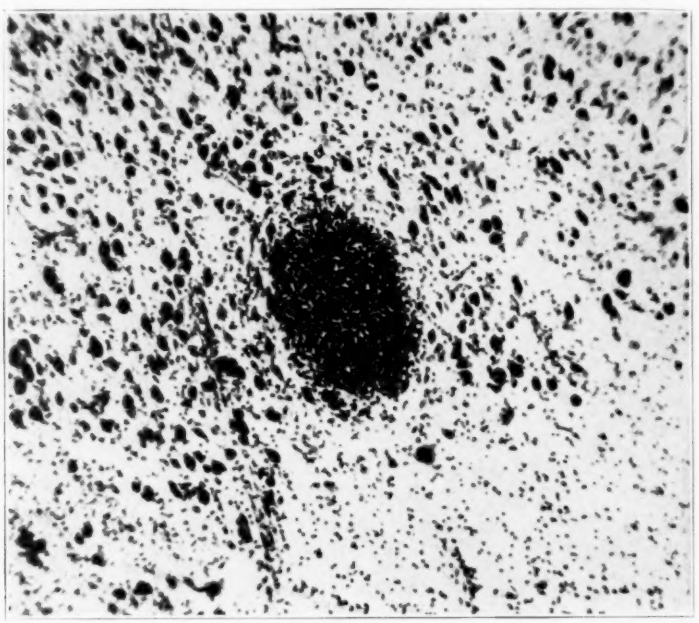

Fig. 2.—Metastatic focal encephalitis in the smaller cell layers of the external geniculate body caused by sepsis following tonsillitis. The lesion consists principally of polymorphonuclear leukocytes and cellular débris. Nissl stain.

Comment.—In the forms of acute encephalitis available for study the external geniculate body showed acute encephalitic lesions in each case, despite the fact emphasized by Spatz [21] that the various forms have a different mode of spreading. Since functional capacity may exist despite considerable pathologic damage in the central visual system, probably because of binocular vision, it is doubtful if these small local-

20. Diamond, I. B.: Changes in the Brain in Pyemia and in Septicemia, Arch. Neurol. & Psychiat. **20**:524 (Sept.) 1928.

21. Spatz,[19] p. 200.

ized lesions give rise to any clinical signs. Blindness in epidemic encephalitis may result from damage to the striate cortex (Scholz [22]).

Meningitis.—Eight cases of meningitis, including epidemic, purulent, tuberculous and syphilitic types, were examined. All except one showed striking pathologic changes.

Of the two patients with tuberculous meningitis, one was blind and the other had marked diminution of vision. Two other patients had dilated fixed pupils, but were too sick to be examined for visual field defects. The changes in the lateral geniculate body probably explain the visual defects. The meningitic reaction did not differ from that elsewhere in the brain, and the type of reaction

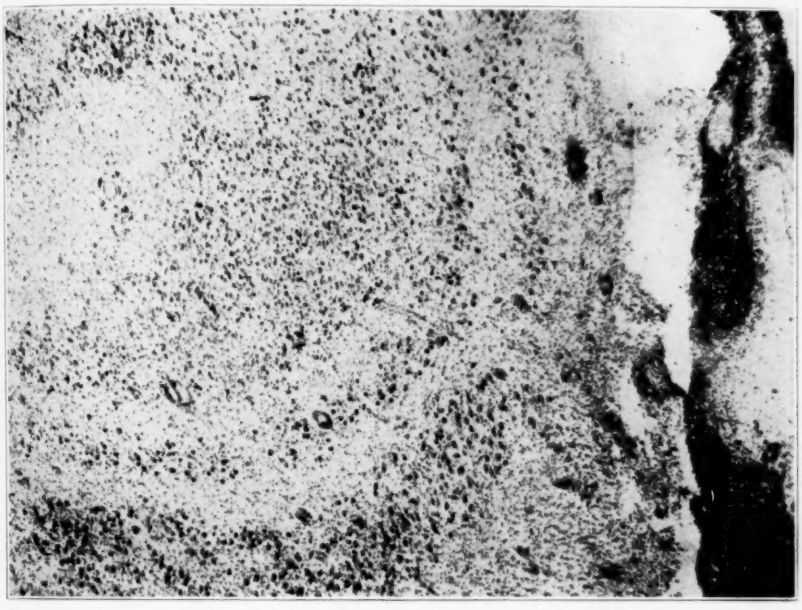

Fig. 3.—Section through the lateral geniculate body in tuberculous meningitis. The severe meningeal vascular and glial reaction can be seen especially in the ventral portion of the ganglion. Above and to the left a circumscribed cell defect is visible. The ganglion cells showed the severe cell disease of Nissl. Nissl stain.

corresponded to the nature of the infection. The pronounced involvement of the cells of the lateral geniculate body seemed to depend on their proximity to the meninges and the subarachnoid space at the base of the brain and the course of the penetrating vessels which provide a means of spread of the infection. If one could assume that toxic products accumulate in the dependent subarachnoid space at the base, it would explain why the anatomic position and blood supply of the lateral geniculate body enter into the question of local vulnerability as they apparently do. Although the meningeal exudate was characteristic of the type of infec-

22. Scholz, W.: Zur Klinik und pathologischen Anatomie der chronischen Encephalitis epidemica, Ztschr. f. d. ges. Neurol. u. Psychiat. **86**:533, 1923.

tion in a given case, the reaction of the ganglion cells and glial elements was quantitative rather than qualitative, and appeared to depend on the course, intensity and duration of the disease.

Figure 3 shows a section through the external geniculate body in a case of tuberculous meningitis. In addition to the usual inflammatory reaction of the meninges and blood vessels, patchy areas of hemorrhage occurred near the meningeal border, and in general the disease was more marked in the ventral ganglion cell layers. However, all the ganglion cells were severely damaged. The glia cells showed progressive changes, the microglia (rod cells) being predominant near the border and the protoplasmic macroglia in the deeper layers. The ganglion cells everywhere showed the severe cellular disease of Nissl, with numerous examples of neuronophagia, glia cell satellitosis or substitution. A circumscribed cellular defect was present in the deeper layers, and some of the ganglion cells lying around a centrally located blood vessel were severely diseased but not destroyed like those in the rest of the cell defect. The blood vessel, in serial sections, was not injured, and did not appear to be the cause of the cell defect. In the cerebral cortex in this case, vascular dependent circumscribed areas of pallor and softening similar to those described by Bodechtel and Opalski [23] and by Wertham [24] were seen. The optic chiasm and tracts were not greatly changed. The origin of the cellular defect in the geniculate body was probably toxic and vascular, since both factors operate to produce changes in the brain in meningitis, according to Spielmeyer,[25] Sittig [26] and others. In other cases of tuberculous meningitis studied the changes differed only in degree.

In purulent meningitis the changes in the ganglion cells and glia cells were of the same type as those already described, only the type of meningeal and vascular exudate being different.

Comment.—Marked visual defects in two cases of tuberculous meningitis depended on the spread of the process along the hilar vessels and damage to the cells of the lateral geniculate body itself, and were not due to damage to the optic tract or to secondary hydrocephalus, as has been emphasized by older authors.[27] Lenz found reports of visual defects in epidemic meningitis without pupillary or ophthalmoscopic findings; he stated that fixed dilated pupils may also result from lesions in the central visual system. The severity of the damage to the lateral geniculate body seen in seven of the eight cases of meningitis studied could readily explain the visual defects produced by that disease.

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23. Bodechtel, G., and Opalski, A.: Gefässbedingte Herde bei der tuberkulösen Meningitis, Ztschr. f. d. ges. Neurol. u. Psychiat. **125**:401, 1930.

24. Wertham, F.: The Cerebral Lesions in Purulent Meningitis, Arch. Neurol. & Psychiat. **26**:549 (Sept.) 1931.

25. Spielmeyer, W.: Pathogenese örtlich-electiver Hirnveränderungen, Ztschr. f. d. ges. Neurol. u. Psychiat. **99**:756, 1925.

26. Sittig, O.: Ueber herdförmige Destruktionsprozesse im Grosshirn und Veränderungen im Kleinhirn bei tuberkulöser Meningitis, Ztschr. f. d. ges. Neurol. u. Psychiat. **23**:511, 1914.

27. Lenz, G.: Zur Pathologie der cerebralen Sehbahn unter besonderer Berücksichtigung ihrer Ergebnisse für die Anatomie und Physiologie, Arch. f. Ophth. **72**:1 and 197, 1909.

Uremia.—Figure 4 was taken from a case of uremia of three months' duration. The patient had decreased vision, but there were no changes in the fundus except narrowing of the lumen of the blood vessels. The ganglion cells showed several varieties of pathologic changes. The nuclei in some cells were dark-stained, shrunken and irregular; in other cells the nuclei were large, with distinct granules and large vacuolated nucleoli. The Nissl granules in some cells were clumped, and in others were completely dissolved. Some of the cells showed central chromatolysis and shrunken nuclei, giving an atypical picture of axonal reaction. Many ganglion cells showed a pronounced degree of pigment atrophy, fatty sclerosis or simple shrinkage. No neuronophagia or satellitosis was seen.

Both progressive and regressive changes of the fixed glia elements were present, and the glia cells were seen in increased numbers. The regressive changes had

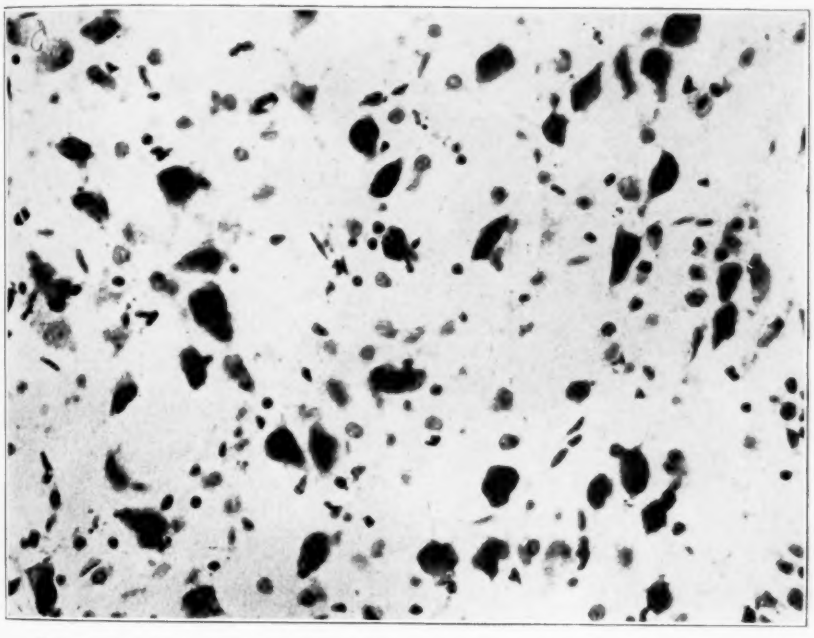

Fig. 4.—Cell changes in the external geniculate body in uremia. The protoplasmic glia cells can be seen; changes in the ganglion cells are less distinct. Some shrunken cells can be seen. Nissl stain.

occurred secondary to the progressive changes. Small clusters of the protoplasmic form of both the macroglia and the rod cells were also seen. There was an increased amount of pigment in the endothelial cells of the small blood vessels.

Comment.—The diffuse degenerative changes in the nerve cells accompanied by lively protoplasmic growth of the glia cells, which were seen singly or in small groups, correspond to similar changes found widely distributed in the brain by many workers.[28] It is difficult to

28. Hartmann-Schrottenbach: Endogene Vergiftungen, in Lewandowsky, M. H.: Handbuch der Neurologie, Berlin, Julius Springer, 1910-1914, vol. 3.

determine how much of this change was due to accompanying vascular disease and how much to the uremia itself. In the presence of the insignificant changes in the fundus oculi it is possible that the changes in the lateral geniculate body contributed to the visual disturbance that was present clinically. Willbrand and Saenger [29] studied cases of sudden blindness in uremia. They concluded that visual defects may be due either to a localized lesion in the central visual system or to a functional defect. The visual fields may show no defect when the blindness is developing. Further clinicopathologic studies of the central visual system in uremia are needed to confirm the views given here.

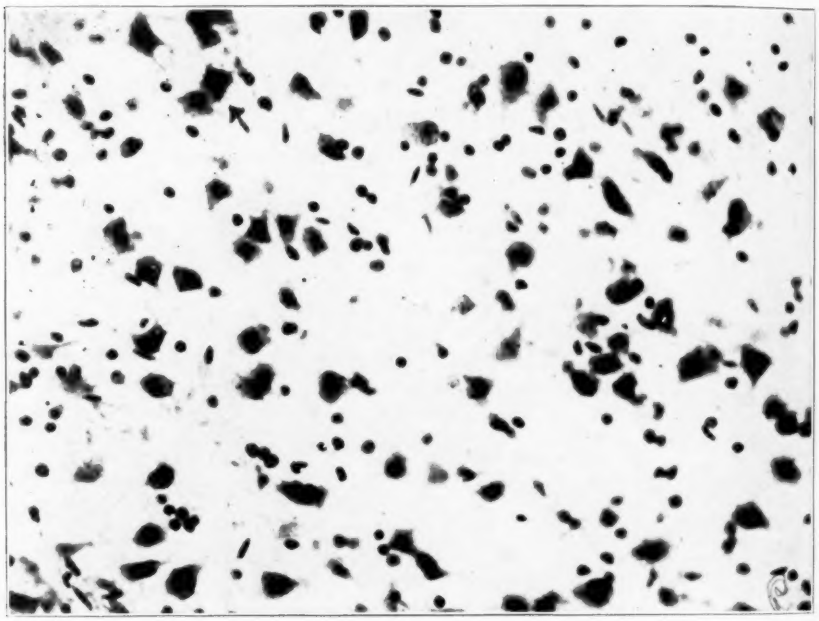

Fig. 5.—Severe disease of the ganglion cells in the lateral geniculate body in status epilepticus. The cell indicated by the arrow shows impregnation of the Golgi net. Nuclear changes in the ganglion cells and small clusters of glia cells can be seen. Nissl stain.

Epilepsy.—In nine cases of epilepsy the changes in the lateral geniculate body were compared with those in the cornu ammonis, which is the site of relatively constant changes in both symptomatic and genuine epilepsy.

The ischemic cell change, so frequent in Sommer's sector of the cornu ammonis, was not seen in the cells of the nearby lateral geniculate body, at least in the typical form. In six of the cases the typical ischemic cellular change in Sommer's

29. Willbrand and Saenger, quoted by Lenz,[27] p. 14.

sector was seen. In the lateral geniculate body three of the six cases showed the severe cell disease of Nissl; in the remaining three only pigment atrophy was present. Figure 5 illustrates the condition in the geniculate body in a patient who died in status epilepticus. The ganglion cells show severe cellular disease. The nuclei were shrunken, darkly stained and irregular in shape. The cytoplasm was homogeneous and undergoing vacuolation and disintegration. Impregnation of the Golgi net occurred in many cells. There was a moderate increase in all types of glia cells. A good proportion had hyperchromatic nuclei and were undergoing regressive changes. Small groups of macroglia cells (*Gliarasen*) were seen.

Comment.—The type of change in ganglion cells described is similar to that observed by Scholz [30] in the cortex in status epilepticus. He attributed the change in the cortical cells to ischemia, which was secondary to a functional vasomotor spasm of the cortical vessels. It has already been mentioned that the typical ischemic cellular change seen in the cornu ammonis did not occur in the lateral geniculate body, but the type of severe cellular disease described may be ischemic in origin. The relationship of such changes to the complex visual phenomena associated with epilepsy is, of course, unknown. Interesting theoretical questions in that direction are suggested by these observations.

CELLULAR CHANGES ASSOCIATED WITH CELLULAR DEFECTS IN THE EXTERNAL GENICULATE BODY

It is beyond the scope of this paper to consider the representation of the retina in the central visual system. That subject has been clarified by Minkowski,[1] Henschen,[31] Brouwer,[3] Putnam [4] and others. Only the type of change in the ganglion cells in the external geniculate body produced by lesions in the different parts of the visual system will be considered.

Destruction of the Optic Nerve or Tract.—Cellular degeneration in the external geniculate body resulting from destruction of the optic nerve or tract is a secondary atrophy of disuse. No examples of this type of atrophy were found in the material studied. However, Hechst [32] described the cellular changes secondary to peripheral blindness, which consist essentially of shrinkage of the ganglion cells, with pigment atrophy and poor staining of the dendrites with basic dyes. The nucleus resists the process for a time, but as the cell undergoes atrophy it finally shrinks until only a small dark homogeneous disk is left of the cell. The alternating atrophy of Minkowski produced by such lesions is well known.

Defects Involving the Optic Radiations.—A lesion in the course of the optic radiations produces primary cellular degeneration of the lateral geniculate body.

30. Scholz, W.: Ueber die Entstehung des Hirnbefundes bei der Epilepsie, Ztschr. f. d. ges. Neurol. u. Psychiat. **145**:471, 1933.

31. Henschen, S. E.: Zur Anatomie der Sehbahn und des Sehzentrums, Arch. f. Ophth. **117**:403, 1926.

32. Hechst, Béla: Ueber das Verhalten der äusseren Kniehöcker und der Sehrinde bei einseitiger peripherer Blindheit, Arch. f. Psychiat. **100**:19, 1933.

This reaction apparently may occur rapidly, but the exact time needed for complete cellular degeneration in man is probably not known. In old lesions the smaller cells disappear completely. This is shown in figure 6, which was taken in a case of diffuse sclerosis. This is an old lesion, as the patient's blindness had been present for over a year before death. Both the axis-cylinders and the myelin sheaths of the fibers of the optic radiations were destroyed. In the lateral geniculate body it can be seen that the small and medium-sized ganglion cells have almost entirely disappeared, but a part of the large ganglion cells of the two large cell layers, as well as the scattered large cells in the deeper layers, are still intact. These large cells showed either pigment atrophy or a loss of Nissl granules and homogeneous cytoplasm. The nuclei were large, clear and swollen, or dark-stained and shrunken. The fixed glia elements were markedly increased in number, with

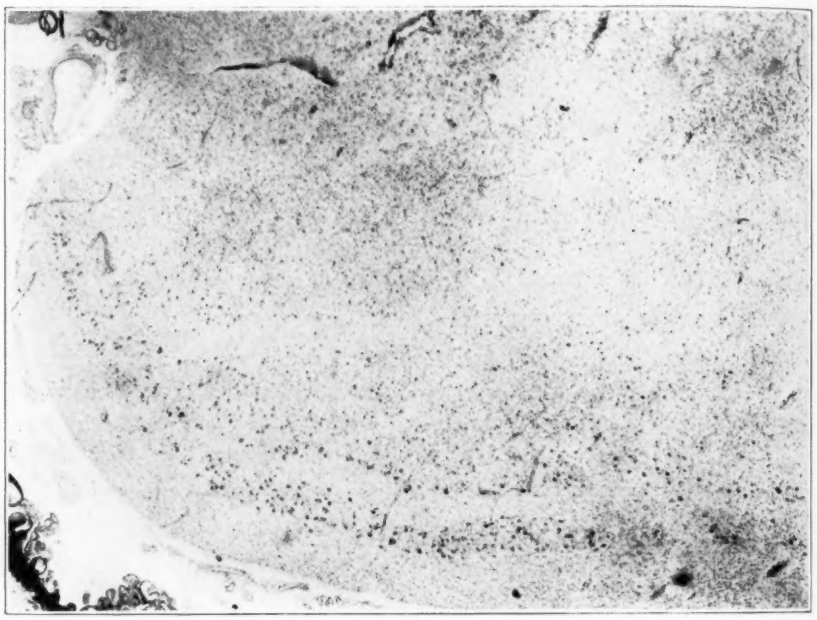

Fig. 6.—Extensive cellular defect of the external geniculate body secondary to the destruction of the striate cortex and optic radiations in a case of diffuse sclerosis. Note the persistence of part of the large ganglion cells, especially in the ventral large cell layers. Nissl stain.

the presence of protoplasmic forms such as the so-called *gemästete* glia cells and gitter cells, especially around the blood vessels.

The persistence of some of the large ganglion cells after the smaller cells are destroyed is well known. Lenz [33] thought that the large cells subserve a motor optic function and hence do not send fibers to the striate cortex. However, Henschen maintained that the large cells mediate impressions between the retina and the calcarine area. Brouwer [34] recognized that the large cells are destroyed in

33. Lenz,[27] p. 105.

34. Brouwer, B.: Ueber die Sehstrahlung des Menschen, Monatschr. f. Psychiat. u. Neurol. **41**:129 and 203, 1917.

some cases, but are preserved in others, although they finally degenerate if the lesion is of long duration. He agreed with von Stauffenberg that the persistence of the large cells is probably because they are more resistive to retrograde degeneration than the smaller cells.

Changes Secondary to Vascular Lesions.—The cellular defect shown in figure 7 was produced by an extensive arteriosclerotic cerebral softening of about two months' duration. Clinically, the patient had homonymous hemianopia on the right and hemiagnosia on the left. There was involvement of the insular region on the right which began at the anterior end of the fossa of sylvus and involved a part of the lower parietal, the upper temporal and the anterior occipital cortex; in other words, the cortex supplied by the middle cerebral artery was involved. Destruction of a part of the internal sagittal stratum in the temporal lobe was

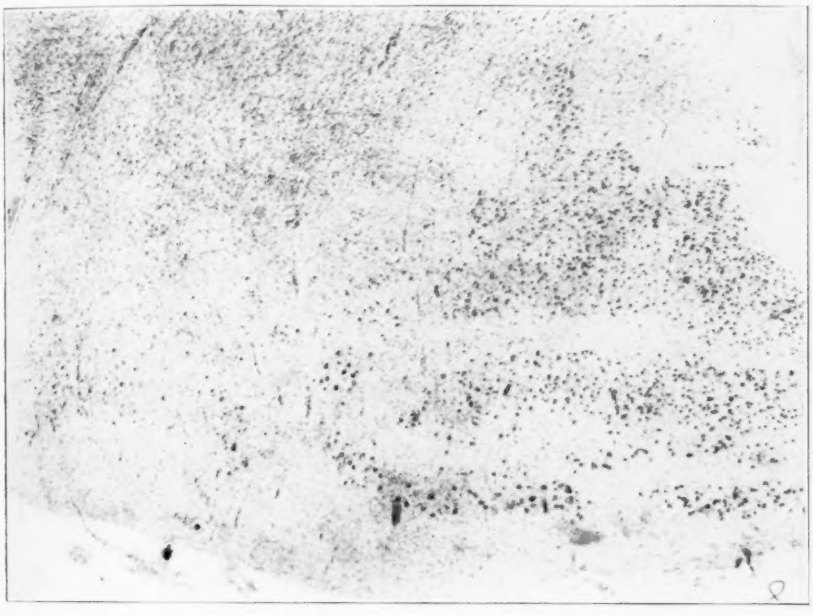

Fig. 7.—Cellular defect of the medial portion of the lateral geniculate body in cerebral arteriosclerosis, with softening of the temporal lobe. The optic radiations were partially destroyed. Nissl stain.

responsible for the ganglion cell defect in the lateral geniculate body. Here the cellular defect was limited to the medial half of the ganglion, and the cells had undergone almost complete degeneration. Some of the large ganglion cells remained intact, but showed high grade pigment atrophy and hyperchromatic nuclei which were undergoing karyorrhexis. Individual cells showed homogeneous cellular change. Pigment rests were seen. The glia cells showed an abundance of protoplasmic forms, many with hyperchromatic or pyknotic nuclei. No mobile glia cells were seen.

In figure 8 the arteriosclerotic softening had extended directly into the external geniculate body from Wernicke's zone and produced a cellular defect in the lateral portion of the ganglion. In this case the softening was of only five weeks' dura-

tion, and was limited almost entirely to the white matter. The defect apparently resulted from destruction of the emerging optic radiation fibers. The changes in the ganglion cells were identical with those in figure 7. The large ganglion cells were spared to a considerable degree. Fresh protoplasmic macroglia and gitter cells were seen in large numbers.

Comment.—It is obvious that the changes in the ganglion cells in the external geniculate body resulting from lesions in the course of the visual pathways have the same histologic stamp. As emphasized in other conditions, pigment atrophy occurs before complete cellular disintegration. A case was found of arteriosclerosis of the very small hilar

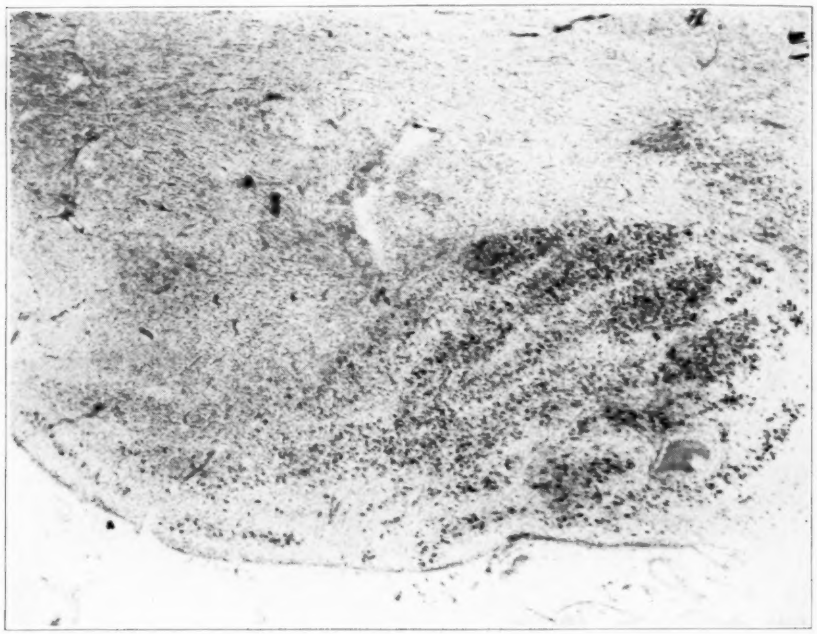

Fig. 8.—An arteriosclerotic softening in Wernicke's zone produced the destruction of the emerging fibers of the optic radiations leading to the cellular defect in the lateral geniculate body. A part of the last dorsal smaller cell layer is apparently directly destroyed. The type of change in the ganglion cells in figures 5, 7 and 8 is similar to that shown in figure 6.

vessels without destruction of the optic tract or optic radiation. In this case only diffuse dropping out of cells and severe pigment atrophy were seen. This case possibly illustrates the loss of cells from simple reduction in the blood supply.

SUMMARY

1. The cellular changes in the external geniculate body in a wide variety of toxic, infectious and degenerative conditions were studied.

2. This structure is not particularly sensitive to changes post mortem when compared with other parts of the central nervous system.

3. In severe infections and toxemias the ganglion cells undergo swelling and chromatolysis and especially the change described by Nissl as severe cell disease. The most common change in the ganglion cells was pigment atrophy. Cells undergoing degeneration from interruption of the visual pathways or from subacute and chronic degenerative diseases develop an excessive accumulation of lipoid pigment.

4. The reaction of the glia cells was varied, seeming to depend on the intensity and duration of the disease process.

5. Some findings of particular interest in encephalitis, meningitis, uremia and epilepsy are described.

I am indebted to Professor Spielmeyer and to Professor Scholz of the German Research Institute for Psychiatry, Munich, for their help and criticism in the preparation of this paper.

EXPERIMENTAL LESIONS OF THE PYRAMIDAL TRACT

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The experiments reported here represent an attempt to produce lesions in the corticospinal or pyramidal tracts without damage to other motor systems. This cannot be attained by operative lesions on the motor cortex, for there descend from here many other efferent fibers besides the pyramidal, as Mellus [1] long ago demonstrated. Lesions in the cord are equally unsatisfactory, for the pyramidal tract in this location can hardly be completely interrupted without damage to the rubrospinal system, although Ranson, Muir and Zeiss [2] reported the case of one cat in which they thought that this might have been attained. The most satisfactory site is in the medulla, where the pyramids lie exposed on the ventral surface for a short distance above the decussation. Since not all of the fibers cross the midline, it is necessary to cut both tracts in order to remove completely pyramidal innervation from even one side of the body. Section of only one pyramid or of the pyramidal decussation leaves intact in the dog (Muratoff [3]), cat (Boyce [4]) and monkey (Sherrington [5]) a small but definite uncrossed tract in the lateral column, and in anthropoid apes one in the anterior column as well. Operations in the region with survival long enough for adequate studies of degeneration are few, and in most of these the physiologic findings are incomplete. These will be discussed in a later section.

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A preliminary report of this study has been published (Lesions in the Pyramidal Tracts in Cats, Proc. Soc. Exper. Biol. & Med. **31**:68, 1933).

1. Mellus, E. L.: Experimental Degeneration Following Unilateral Lesions of the Cortex Cerebri in the Bonnet Monkey (Macacus Sinicus), Proc. Roy. Soc. London **58**:206, 1895.

2. Ranson, S. W.; Muir, J. C., and Zeiss, F. R.: Extensor Tonus After Spinal Cord Lesions in the Cat, J. Comp. Neurol. **54**:13, 1932.

3. Muratoff, W.: Secundäre Degeneration nach Zerstörung der motorischen Sphäre des Gehirns in Verbindung mit der Frage von der Localisation der Hirnfunctionen, Arch. f. Anat. u. Physiol. (Anat. Abt.), 1893, p. 97.

4. Boyce, R.: A Contribution to the Study of Descending Degeneration in the Brain and Spinal Cord, Phil. Tr. London, B **186**:321, 1895.

5. Sherrington, C. S.: Note on Experimental Degeneration of the Pyramidal Tract, Lancet **1**:265, 1894.

METHOD

The findings to be reported are based on observations on seventeen cats, twelve with bilateral and five with unilateral lesions of the pyramids above the medulla.

The operative procedure used was that devised by Starlinger [6] and is briefly as follows: The animals were anesthetized with phenobarbital sodium or sodium amytal. A long incision was made in the ventral aspect of the neck in the midline, starting anteriorly as near the symphysis of the mandible as it was conveniently possible to get. After the instruments were passed in between the trachea and esophagus on the one side and the right carotid artery, jugular vein and vagus nerve on the other, the prevertebral muscles were reached. The dissection was then continued forward, and the prevertebral muscles were stripped from the basiocciput between the tympanic bullae. The pharynx was not opened. The periosteum was scraped from the basiocciput, and a trephine opening was made in the bone. The opening was made larger, if necessary, with an ethmoid punch or with fine rongeurs. The dura was slit with great care to prevent injury to the underlying basilar artery, which in the cat lies over the midline of the medulla. The pyramids were then cut with a knife, and after all of the bleeding was controlled, the wound was closed in layers.

The animals were observed for a period varying from five to thirty-two days. Sections of the cord and brain stem were made at various levels and studied by means of the Marchi method and iron hematoxylin.

EXPERIMENTAL RESULTS

As the effects of the anesthesia used did not pass for a number of hours, the findings to be reported began with the second day. At this time only one animal, the first of the series (cat 23), was unable to walk, while some of the others showed a certain amount of staggering, slipping of the feet on a smooth floor or unsteadiness in standing. The remainder by this time walked at least moderately well, and those that showed the grosser defects initially were practically over them on the following day. All of the animals, however, showed a number of deficiencies which will be described under the headings of placing reactions, hopping reactions, extensor tonus, flexion activity, posture and movement.

Placing Reactions.—The placing reactions were investigated by the following methods:

1. Light touch applied to the dorsum of the foot by the methods of Munk and Rademaker. In Munk's method (*Berührungsreflexe*) the animal is suspended by the neck, while the paws are stroked lightly with the thumb and forefinger. The normal response is flexion of the limb. In Rademaker's test (one of the group of *Stehbereitschaft*) the animal is supported ventrally while the dorsum of the foot is brought gently against the edge of a table, care being taken that the animal does not see the contact taking place. Normally the foot is promptly lifted and set down on the table. Of the two procedures, Rademaker's method gives, as a

6. Starlinger, J.: Die Durchschneidung beider Pyramiden beim Hunde, Neurol. Centralbl. **14**:390, 1895; Die Durchschneidung beider Pyramiden beim Hunde, Jahrb. f. Psychiat. u. Neurol. **15**:1, 1897.

rule, the greater response and is therefore the more valuable in the early postoperative stages to detect minimal responses. On the other hand, in the later stages, when the limbs are quite active, and particularly after unilateral operations, Munk's method, by producing a smaller response, is sometimes more useful in determining the differences in the two sides.

2. Rademaker's method of touching the tail or rump against the edge of a table to obtain the reactions in the hindlegs, and touching the snout to obtain the reactions in the forelegs. In the cat, touching the tail is less effective than it is in the dog, so that contact to the rump is necessary for the first test. Normally, the hindlegs flex, and the feet are placed on the table. The snout test is rarely satisfactory without a preliminary blinding of the animal, a procedure not used in this series.

3. The visual placing reaction. The animal is slowly lowered, head down, to the top of the table or floor, when normally the forelegs reach forward and are accurately placed on the surface below.

4. Ladder and track tests. The ladder used in these experiments had flat rungs, three-fourths inch (1.9 cm.) in width and placed 1½ inches (3.8 cm.) apart. Two tracks were used; one was 1 inch (2.5 cm.) and the other 3 inches (7.6 cm.) in width. All were several feet long and were placed horizontally about 4 feet (1.2 meters) above the ground. The normal animal can walk these without difficulty and can turn on even the narrowest track; if the feet are pulled off the track by the examiner they are replaced accurately and without difficulty.

In the animals on which operation was performed the simple contact reflexes of Munk and Rademaker were practically always abolished initially in all four feet after unilateral and bilateral operations (fig. 1, *A* and *C*). In the cases of unilateral lesions the homolateral foreleg recovered completely within a few days, while the homolateral hindleg improved more slowly and in some cases showed a permanent defect. In all of the limbs in the cats with bilateral lesions and in the contralateral limbs in those with unilateral lesions, the defects were more severe. In these, movements could usually be elicited in from four to twenty-one days, although as a general rule at the end of the period of observation a certain defect, in that the reactions were less extensive and less brisk than in the normal animals, was apparent. In a few cases (cats 31 and 33) no recovery was noticed, and in a few others (cats 28 and 30) it was unusually rapid and appeared to be complete.

The response to touching the rump against the edge of the table was initially reduced, so that the animals could frequently be placed on the table in a sitting position, with the legs extended forward and downward (fig. 1 *B*). As recovery took place this position of extension gave way to one of flexion, and later the legs were vigorously flexed and placed on the top of the table. The rate of recovery more or less paralleled that of the previous test.

The visual placing reactions also showed a certain defect. It was most easily seen in the cases of homolateral lesions in which the contralateral forelimb was either not advanced at all or only considerably after its fellow (fig. 1 *D*). The disorder in the visual reactions was, however, less in amount than that affecting the contact reactions. Thus in some cases of unilateral lesions (e. g., cats 40 and 41) in the early days, when the contact reflexes were absent in the forelegs, the visual reactions were obtainable, although they were more defective in the contralateral side. In a few instances the combined effect of vision and contact of the feet and nose produced a reaction initially, so that when the animal was gradually lowered to the floor it made no effort to place its feet and fell over in a heap on its side (fig. 1 *E*). The more marked manifestations disappeared within a day or two,

but a slight retardation persisted for a long time. In the animals with bilateral lesions it was hard to tell when the "normal" had been reached, but in the cases of unilateral lesions a slight defect was seen for a long time in that, in response to visual stimuli, the homolateral and more normal limb was always placed on the table ahead of its fellow.

In the tests with the ladder and track the placing defects were easily apparent initially (fig. 1*F*). The animals were disinclined to walk along the apparatus. When they moved, progression was slow and uncertain, and the legs tended to be misplaced so that they fell between the rungs or off the track. Once off, they were returned with difficulty or were simply left hanging down. In the later

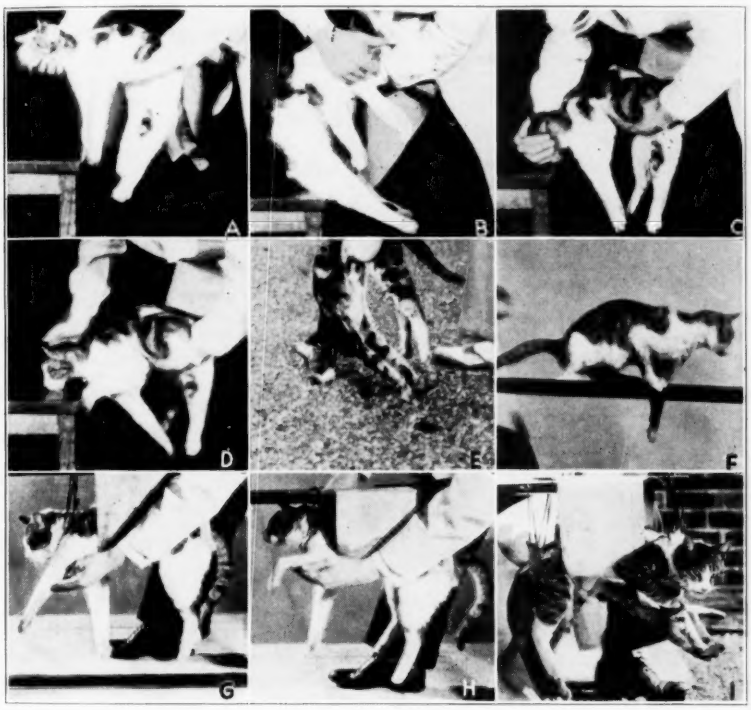

Fig. 1.—*A* (cat 33), absence of the contact reaction in the forelegs. *B* (cat 33), absence of response in the hindlegs from contact of the rump. *C* (cat 33), absence of response in the foreleg from contact of the nose, with the vision excluded. *D*, same as *C*, with vision included; a reflex is present in the left leg and absent in the right. *E* (cat 25), absence of response, with combined effects of contact of the feet, nose and vision. *F* (cat 35), test on the narrow track; the left foot has fallen off and not returned. *G* (cat 35), resistance to passive flexion at the elbow. *H* (cat 35), disappearance of resistance to flexion when the skin of the neck and the upper part of the back is grasped. *I* (cat 40), increased resistance to flexion, a positive *Stütz* reflex in the left forelegs from a right pyramidal lesion.

stages there was no spontaneous misplacement, but turning on the narrower track was frequently carried out with difficulty. Still later, the only observable defect

was a hesitating, uncertain replacement of a foot pulled off the track by the examiner. Finally, at the end of the observation period, little or nothing could be observed.

Hopping Reactions.—The hopping reactions were examined in the following ways:

1. One leg was tested at a time, after the method of Rademaker. The animal was held horizontally over a floor covered with a rubber or cord mat. One foot only was allowed to rest on the floor, and the body, which was held horizontally, was moved in various directions. The important factors in the response are the distance the body must be moved to initiate a hop and the character of the hop. Normally a hop is most readily induced by movement of the body forward and laterally and is less easily produced by movements in other directions. In some cases with mesial or backward motion, the legs may not hop but are merely dragged.

2. The animal was made to walk on the two forelegs or the two hindlegs only. In testing the forelegs the hindquarters were held higher than the forequarters, and in testing the hindlegs the body was almost vertical. Normally the forelegs step quickly and easily. The hindlegs are more awkward, but the leg is flexed, moved forward and then set down.

Of the animals operated on, seven with bilateral lesions and all of those with unilateral lesions were tested for hopping on one leg. All showed initial defects. There was a retardation or delayed reaction, so that a larger body movement was required to produce a hop, or in some cases no hop could be evoked. The limbs were sometimes dragged or imperfectly lifted, so that the toes scraped along the floor, or the movements were simply awkward and heavy. The defects were greater in the hindlegs than in the forelegs and were greater in the case of movements in the mesial and posterior directions, as compared with those in the forward and lateral directions. If one bears in mind, however, the differences in the normal animal, it is difficult to say that the operation produced greater deficiencies in hopping in any particular direction. After unilateral operations the contralateral legs were most involved, the homolateral hindleg was affected to a lesser degree, and the homolateral foreleg, not at all. To some extent, therefore, the hopping reactions were disordered less initially than were the placing reactions, which were absent at first in the same cases in all four limbs.

A certain amount of recovery in the hopping reactions usually took place, with a tendency for the forelegs to improve more rapidly than the hindlegs. Nine animals lived two weeks or more, and of these, two (cats 28 and 30) showed complete and rapid recovery, three (cats 39, 40 and 41) showed slower progress with probably complete return to normal, two (cats 31 and 32) showed recovery in the forelegs but not in the hindlegs, and three (cats 33, 35 and 38) showed a defect at the end in all four legs.

Walking the animal on the hindlegs was found to be a valuable method of bringing out the sort of deficiencies previously noted. The defect was most easily observed in the contralateral leg after unilateral operation. When the trunk was raised to the vertical position, the affected limb stood extended stiffly forward, frequently not touching the floor, while the other leg bore the weight of the body and was slightly flexed. When the animal was moved forward, the stiff leg was dragged or moved along awkwardly, with little flexion as compared with the other leg. Improvement occurred here as elsewhere, but walking on the hindlegs not infrequently showed defects when hopping on one leg appeared to be normal.

The results of making the animals walk on the forelegs added little or nothing to what had been learned from other tests.

Extensor Tone and Flexor Activity.—Extensor tone was tested by the following methods:

1. The animal was suspended in a canvas sling which passed under the ventral aspect of the thorax and abdomen and left the feet hanging free. This method was a modification of one described by Ranson, Muir and Zeiss,[2] which consisted of a wooden frame to which the supporting canvas was attached. The frame was rectangular, measuring 1 by 3 feet (33 by 91 cm.) and was supported by four legs about 2½ feet (76 cm.) above the ground. To support the animal Ranson used a sheet of muslin with four holes for the legs to pass through, but it was found extremely difficult to keep an active cat in such a device from struggling and clawing at the cloth. A piece of canvas just wide enough to pass under the abdomen between the forelegs and the hindlegs was found more satisfactory. To this were attached two narrower T pieces, the stems of which passed between the forelegs and hindlegs, while the cross-pieces passed around the neck and tail. Two brass rods were attached to the sides of the body piece, and cords were fastened to the ends of the T pieces. In use, the sling was put around the standing animal, and the whole was quickly lifted and snapped onto cup-hooks set in the top of the frame. The position of the animal in the frame was such that it could not reach the woodwork with its claws. The cats seemed to object to this apparatus less than the other supporting devices, and even active cats could usually be kept in it with a little encouragement. The animals on which operation was performed initially gave no trouble, and progress in recovery could frequently be estimated by their general behavior in the sling.

When the normal control was supported in the sling, the forelegs and hindlegs behaved somewhat differently. The hindlegs at rest were usually partly flexed, and manipulation by the examiner in the form of passive flexion or extension as a rule produced a vigorous active flexion of the leg. There was no resistance in particular to passive flexion. In the forelegs the patterns were more variable. The legs at rest hung straight down or were partly flexed. They were frequently in motion, and then an accurate test of passive movement was difficult to obtain. Passive flexion could at times be easily elicited, while at other times a certain resistance was felt. Passive extension did not produce the vigorous movements of flexion so characteristic of the hindlegs.

2. The animal was placed on its back in a trough, with its feet in the air. The normal animal usually resisted being put into this position and struggled a great deal. When the animal was quiet, the hindlegs were usually flexed and resisted extension. At times, particularly after attempts to rise, the hindlegs were found to be extended, but they usually flexed readily in response to any passive manipulation. The forelegs were sometimes variable in position, but they were usually more or less flexed and showed little or no resistance to passive flexion.

3. The animal was held horizontally by the nape of the neck and the tail, with the feet hanging down. Normally the hindlegs were partly flexed and showed no resistance to passive flexion. The forelegs were as before—variable.

4. The animal was held by the nape of the neck only, so that the trunk hung vertically downward and the head was bent slightly forward. Normally, in this position all four legs were flexed, or the hindlegs were extended, perhaps showing a certain resistance to flexion.

5. The animal was held with the examiner's hand under the thorax, so that the anterior part of the body was horizontal, while the posterior part hung vertically

down. In this position, the tendency toward extension of the hindlimb was further increased over the preceding test, probably through the functioning of the tonic reflexes of the neck.

In all of these postures the resistance to passive flexion was determined (*a*) by gentle pressure or tapping applied to the limb below the knee and elbow in such a way as to produce flexion at these joints only and (*b*) by upward pressure on the pads of the feet after the manner used in eliciting the *Stütz* reflex. In the sections to follow, unless the *Stütz* reflex is designated, resistance to passive flexion was determined by the first method.

Finally, in addition to the preceding specific tests, further evidence of muscle tonus was deduced from the posture and gait.

Of the five positions described, the most satisfactory was the first, with the animal in the ventral position in the sling. The animals could be kept quieter and less disturbed in this position than in some of the other positions, and it was easier to obtain the identical posture from day to day. The reactions obtained in this apparatus will be described first, and any differences obtained in other positions will be noted later. The possible objections to the sling method will be discussed in a subsequent section.

In the sling, three abnormal findings were observed in the early postoperative days: (1) The limbs were extended in position; (2) they resisted passive flexion, and (3) the normal resistance to passive extension was generally reduced.

The resistance to passive flexion in the hindlegs was usually definite and varied from a slight amount to such a high degree that in some cases the knee could hardly be bent at all. In the bilateral operations both hindlegs were affected, and in the cases of unilateral lesions the contralateral leg was chiefly involved, although the homolateral leg was usually affected to some extent. One animal with a unilateral lesion (cat 36) had initial hypotonia on the contralateral side and slight hypertonia on the homolateral side, but in this animal the pathologic findings were somewhat different from the others. The nature of the resistance varied. In some cases, when the pressure was released, the limb quickly sprang back to its original extended position. In others the limb was more plastic, and although it resisted flexion, it took, within limits, any position imposed on it. The amount of resistance could frequently be altered by various means. It could at times be reduced by firmly grasping a large area of skin over the lower part of the back and sacrum. Repeated flexions sometimes had the same effect. On the other hand, it was often considerably augmented after a period of struggling. The posture of the limb more or less paralleled the degree of resistance to flexion, being extended when the degree of resistance was high and flexed when it was low.

With the resistance to passive flexion in the hindlegs there went a reduction or absence of resistance to extension and a general reduction in flexor activity. The latter showed itself clearly in the cases of unilateral lesions; while the homolateral leg participated in struggling movements, the contralateral leg remained quiet. The diminished resistance to extension did not necessarily parallel, and therefore cannot be regarded as being entirely dependent on, the increased extensor tonus, for frequently it was considerably or totally reduced, with only a slight degree of extensor tonus, and the cat which showed hypotonia had also a reduced resistance to flexion in the same limb.

In the forelegs there was a definite and frequently a high grade resistance to flexion, which was sometimes less than, but perhaps more commonly greater than, that found in the hindlegs of the same animal. Since, however, the normal control sometimes showed a certain resistance to the same procedure, the actual increase

as a result of the operation was difficult to determine. In the animals with unilateral lesions the contralateral forelimb showed a constant resistance. The homolateral limb sometimes did and sometimes did not show resistance, and when the resistance was present it was usually less in amount than in the contralateral leg. It could frequently be reduced if one grasped the skin of the back over the scapulae (fig. 1 *G* and *H*). Resistance to extension could not be used as a test in the forelimb, for the normal control usually showed little of it. In the animals with unilateral lesions the contralateral forelimb took less part in the struggling movements than did the homolateral forelimb.

In the dorsal position in the trough, the animals showed initially, as a general rule, much less resistance to passive flexion of the hindleg than they did when placed ventrally in the sling, and in some cases little or no resistance could be made out. There was a definite diminution in the normal resistance to extension, and the leg frequently remained in the extended position when pulled out, a thing which rarely occurs in the normal control. Furthermore, passive flexion of the head resulted at times in extension of the hindlegs, a rare occurrence normally with passive, although it may be present with active, flexion. In the forelegs the responses were variable, and since they were also variable in the normal control, direct comparisons were difficult. In only a few instances, therefore, was the diagnosis of a definite increase in resistance to flexion in these limbs justified.

The findings in the remaining test postures did not materially add to the knowledge obtained by the first two methods. The normal animal, when held under the thorax with the head projecting forward and the trunk downward, as already noted, showed a certain tendency toward an extensor position of the hindlimbs. In the animals on which operation was performed this tendency was much increased, and the amount obtained was frequently greater than that obtained in any other position. It could sometimes be reduced by upward pressure on the rump.

A certain amount of recovery in these functions during the period of observation almost invariably took place. The limbs changed from the extensor to the flexor posture in the test positions, the resistance to flexion gradually declined, and the resistance to flexion and flexor activity increased. The resistance to flexion, as tested ventrally in the sling, in some cases disappeared during the period of observation, while in others a certain amount was present to the end. In the dorsal position in the trough it ceased being apparent much earlier than it did in the sling, and it could frequently be brought out by holding the animal under the thorax when other measures failed. The diminished resistance to extension tended to persist longer than did the resistance to flexion when both were tested in the same position.

The resistance to flexion as tested by the *Stütz* method of upward pressure on the soles of the feet (fig. 1 *I*) gave no information not already obtained by other methods. In addition, the results obtained by this procedure appeared to be more variable than those occurring with the other methods, and it was more difficult to make comparisons with it on the two sides. Rademaker [7] called attention to the variability of these reflexes in the cat as compared with those obtained in the dog.

Other indications of an increased extensor tonus were sometimes obtained from the gait and the posture of the limbs. Thus, not infrequently in the first few days, when an animal coughed or sneezed the hindlegs became stiffly extended and slipped forward and laterally. After a series of coughs the animal might be brought down on its rump.

7. Rademaker, G. G. J.: Das Stehen, Berlin, Julius Springer, 1931.

Posture.—In the first few days after operation there were a number of spontaneous postural abnormalities, and certain abnormal postures imposed on the animals by the experimenter were not corrected. Thus, normally, if the paw is turned under by the examiner so that the weight of the body rests on the dorsum of the foot, the foot is quickly lifted and set down properly on the table. In the animal operated on the corrective reflex was lost initially in the affected limbs in all except two animals (cats 24 and 27). In the cases of bilateral lesions it was initially absent as a rule in all four limbs; in the cases of unilateral lesions it was absent in the contralateral limbs, and it was only slightly or not at all reduced in the homolateral limbs. The homolateral hindleg tended to be more disordered in this test than was the foreleg. In all of the cases recovery eventually took place, and the animals appeared to be normal as a rule in from four to twenty-one days. In a few animals at the end of two to three weeks the hindlegs were still affected to some extent, although the forelegs seemed to have recovered. It appears, therefore, that the hindlegs were more involved than were the forelegs, but before accepting this conclusion one more observation must be made. Cats are better able to shift their weight so as to balance on one foreleg than on one hindleg; hence in this sort of test the affected hindleg usually receives more weight than does the affected forelimb. The results of tests of the forelimbs and hindlimbs are, therefore, not directly comparable, and the foreleg may therefore appear to be less involved than it really is.

Spontaneous postural abnormalities of a marked type were observed in a number of animals on the second day. Thus the forelegs sometimes became crossed, the hindlegs slid out in various directions, or the animals stood with the foreleg flexed at the wrist and extended at the carpometacarpal joint. The tendency to assume these curious positions with great frequency lasted as a rule no longer than a day, and a number of animals never showed them. The sliding forward of the hindlegs with coughing has already been noted.

Spontaneous postural disorders of a smaller sort, however, were seen in all of the animals. Thus, there was a tendency to leave the feet in the walking posture after stopping, with one foot of each pair far behind its mate. The toes of the posteriorly directed foot might then be much overextended, much more than enough to incite a forward step or hop in the normal animal; yet in these animals the posture might be maintained for long periods. A defect of a similar nature was seen when the animal was turning; at this time the pivoting leg sometimes became twisted to an abnormal degree before it was moved. These phenomena showed improvement with time, but not infrequently a trace of them was still present at the end of the period of observation.

Of the other postural abnormalities, there was a tendency, only inconstantly present, to stand with the hindlegs abducted and sometimes inwardly rotated. This made the ankle point out and the toes in and gave to the animal a curious bow-legged appearance as seen from the rear. Less frequently the forefeet appeared to be unduly flattened on the floor. A peculiar posture, found at times in the cases of bilateral lesions and less commonly in the cases of unilateral lesions, was one in which the animal squatted with the entire length of its hindfeet from the toe to the ankle on the floor, although the rump was only partly lowered, the whole giving a sort of kangaroo appearance. It appeared sometimes after prolonged standing, sometimes after walking, and in some cases the animal began to "settle" while slowing down preparatory to a stop, so that one observer was led to remark, "He walks sitting down." All of these postural abnormalities improved with time, the kangaroo effect usually rapidly and completely and the abduction and rotation of the leg more slowly and sometimes incompletely.

Movements.—The initial gross disorders of movement in some of the animals have already been noted. Only one was unable to walk on the second day. A certain number of others showed such gross disorders of movement as severe staggering or slipping of the feet on a smooth floor. The others walked fairly well from the beginning, and those that showed the greater deficiencies were usually over them within a short time.

The most constant disorder of the gait itself was a certain degree of stiffness in the hindlegs. It varied in grade in the different animals, being absent in some. In the more severe forms the leg, on being brought forward, swung laterally, describing the arc of a circle, and would then often be set down in the toed-in position previously described. The difficulty improved so gradually that it was difficult to say when the normal state was reached. Thus in some of the animals, in the later stages, it was hardly possible to say that the legs were stiff, but only that the motions seemed less smooth, and that they were more even and regular and showed little of the easy variability so characteristic of the normal control.

In the forelegs, disorder in walking, apart from the initial disturbances previously noted, was only infrequently observed. In a few cases the motions seemed plodding and heavy, and the setting down of the foot gave a heavy impact to the scapulae. Scraping of the toes along the floor in the forelegs or hindlegs was observed in only a few animals and in these for only a short time.

Striking features in almost all of the animals initially were a reduction in the amount of activity and a peculiar slowness and deliberation in the movements. In the early days the animals tended to move relatively little without prodding, although when they started little seemed wrong with the mechanics of the gait itself. The unhurried motion gave the animal a dignified, sedate appearance as it walked around. These peculiarities gradually improved with time; but even at the end of two or three weeks few showed the rapid, vigorous action characteristic of the normal control. A few animals, notably cats 28 and 30, did not show retardation from the beginning.

Finally, many animals showed serious defects in jumping down from a height. The normal control landed at first on the forefeet, and then, after a short interval, the hindlegs lightly hit the floor. The cats which had undergone operation, on the other hand, landed so hard that frequently the breath was knocked out of them. The hindlegs hit the floor too quickly after the forelegs, and then slid forward or bent under the body. In the cases of unilateral lesions, the contralateral legs were chiefly affected, so that the animal tended to fall over to that side. With time the disorder usually almost or entirely disappeared. In one cat (30), however, which on the whole showed a mild group of symptoms, the defect in jumping was one of the few deficiencies remaining at the end of two weeks.

Anatomic Observations.—The brains and spinal cords were fixed in some cases in formaldehyde and in others in Müller's fluid.[7a] The lesions were examined grossly and photographed, and microscopic sections were made at various levels of the cord and brain stem. The sections at the level of the lesion were stained by the Marchi method or by iron hematoxylin. All of the others were treated by the Marchi method.

The lesions were found in all of the cases to involve the pyramidal tract. They were situated at various levels from the midmedulla through the trapezoid body

7a. Müller's fluid is composed of:

Potassium trichromate	2.5 Gm.
Sodium sulphate	1.0 Gm.
Distilled water	100.0 cc.

to the lower portion of the pons. There was usually a certain amount of swelling, in the center of which was a more or less healed scar. The amount of swelling varied in the different cases, and in some it amounted to a small herniation which protruded into the trephine opening of the basiocciput.

Histologically, it was found that the central portion of the pyramids showed as a rule the most severe damage, and from this there radiated a varying amount of gliosis to involve the medial lemniscus which lies immediately behind and in contact with the pyramids. In most cases the lesion involved the entire width of the pyramids, but in some the most mesial or the most lateral portion escaped. In one cat (36) with a unilateral lesion and, clinically, hypotonia of the contralateral limb, the focus was more lateral and involved the lateral portion of the pyramid together with the olivary body. There were frequently a number of small cavities within the main portion of the lesion itself, involving the pyramidal tract or the lemniscus, and in two animals (cats 25 and 31) there were larger cavities

Fig. 2 (cat 25).—Photomicrograph of a transverse section of the lesion. The pyramids were completely destroyed, with damage to the medial lemniscus and raphe; thirteen days' duration. Iron hematoxylin; × 15.

which extended farther back along the median raphe. The cavities were probably the result of injury to the median branches of the basilar artery. Other structures more or less constantly involved were the interolivary fibers when the lesion was low and the trapezoid fibers when it was high.

A descending degeneration was found in all of the cases in the pyramidal tracts. In the bilateral cases both sides were more or less equally involved, and in the unilateral ones the contralateral tract showed most degeneration, although the homolateral one was always involved to some extent. The degeneration could be traced in decreasing amounts to the lumbosacral levels. The amounts varied, dependent partly on the size of the lesion and partly on its duration, which ranged from five to thirty-two days, but the amount was never small. Even the animals which showed the least severe and shortest symptomatology (e. g., cats 28 and 30) had about a 50 per cent degeneration in the pyramidal tracts. Of the animals with bilateral lesions, five (cats 25, 29, 31, 33 and 35) clearly showed a

complete degeneration of both pyramids. The remainder showed moderate-sized or large lesions. Some of the latter may have been complete, but they are excluded from the first group because, for various reasons, such as unsatisfactory slides or short duration of life, it was impossible to state that no fibers escaped. Of the unilateral cases, three were probably complete and two incomplete, one showing

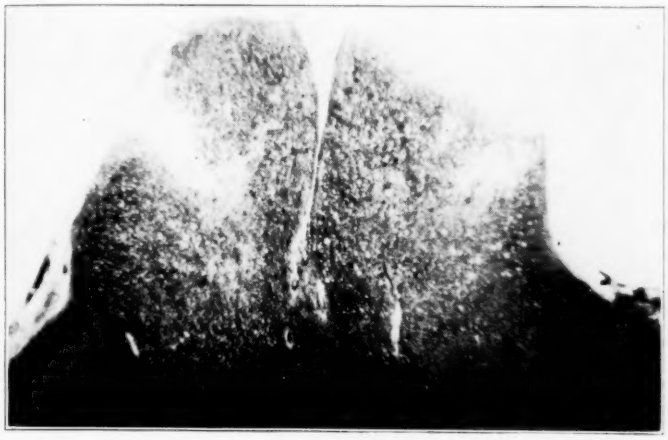

Fig. 3 (cat 31).—Photomicrograph of a transverse section of the lesion. The pyramids are degenerated; duration, twenty-three days. Iron hematoxylin; × 25.

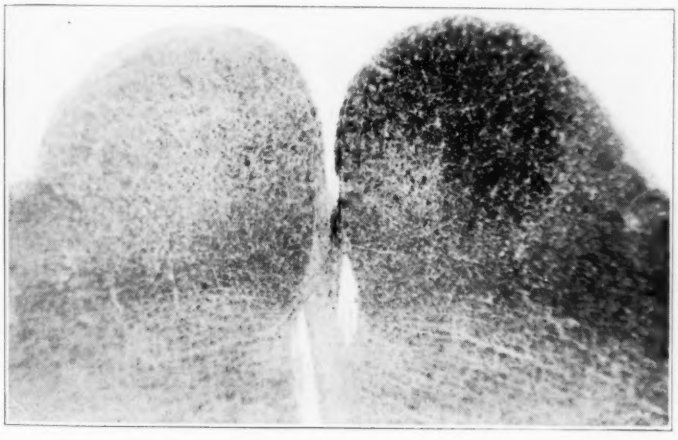

Fig. 4 (cat 41).—The medulla below the level of the injury. The right pyramid, with an incomplete lesion is visible. There are a few scattered degenerated fibers on the left; duration, twenty-one days. Marchi; × 30.

the lateral lesion previously noted. In some animals a descending degeneration, in addition to the foregoing, was found to involve the predorsal bundle in the anterior columns. It was usually bilateral and was rarely of large extent.

An ascending degeneration was found in practically every case which involved the medial lemniscus. It more or less paralleled, but was always much less in amount

than, the pyramidal damage. In the midpons it was usually scattered throughout the lemniscus from its mesial to its lateral extremity, but it was not infrequently concentrated on the ventral border, leaving the dorsal part free.

The essential pathologic changes are, therefore, degeneration of the pyramidal tracts, partial or complete, and associated damage to the medial lemniscus.

COMMENT

The first attempts to section the pyramidal tracts in the medulla were made by Magendie [8] and Schiff,[9] who found little or no observable deficiencies, but gave no anatomic proof of the completeness of their sections. Starlinger,[6] working in von Wagner's laboratory in Vienna, was the first to produce chronic pyramidal preparations with adequate anatomic controls. He sectioned both pyramids above the decussation in six dogs and found a number of symptoms which gradually declined, leaving the animal essentially normal in about two weeks. Soon after the operation many of the animals could run and jump, although some were reported as being slower, more cautious and more deliberate than the normal control. One was reported as jumping down heavily, another as showing marked postural defects, such as crossing the forelegs, and a third as having rolling movements for a few days. Any motor weakness present was in general slight, and spasticity did not appear. The symptomatology was reported as being less severe and of shorter duration than that following removal of the motor areas. Starlinger reported that eventually there were no symptoms which could be attributed to the absence of the pyramids. Histologically, there was degeneration of the pyramidal tracts, more or less complete and traceable for varying distances down the cord. In some cases there was damage to the medial lemniscus, the interolivary region and the median raphe.

In 1900 Schäfer,[10] in a footnote in his "Textbook of Physiology," stated briefly that he was unable to confirm Starlinger's observations in the monkey. In 1910 he described in detail the effect of injury to the pyramids in three of these animals.[11] In two the damage was chiefly unilateral, and in one the pyramids were almost completely severed. Schäfer was particularly interested in "voluntary" movement, and for this employed three tests: (1) the ability to pick up currants from the floor and carry them to the mouth, (2) the tendency of the

8. Magendie, F.: Précis élémentaire de physiologie, Paris, Méquignon-Marvis père & fils, 1839; translated by John Revere, New York, Harper & Brothers, 1844.

9. Schiff, J. M.: Lehrbuch der Physiologie des Menschen; Muskel- und Nervenphysiologie, Lahr, M. Schauenburg & Co., 1858-1859, vol. 1, p. 305.

10. Schäfer, E. A.: Textbook of Physiology, London, Young J. Pentland, 1900, vol. 2, p. 778.

11. Schäfer, E. A.: Experiments on the Paths Taken by Volitional Impulses Passing from the Cerebral Cortex to the Cord; the Pyramids and the Ventro-Lateral Descending Tracts, Quart. J. Exper. Physiol. **3**:355, 1910.

limbs to reach for contact when the animal was swinging or dropped toward a cage or to the floor and (3) the use of the hands in grasping. He considered the second the best test for voluntary movement in the hindlimb and the third variable and rather dependent on the disposition of the animal. Schäfer stated:

> It is noteworthy that after twenty-one days there is manifested a considerable amount of voluntary movement of the paralyzed arm, but none up to twenty-eight days of the corresponding leg, unless its increased facility in walking, climbing and leaping is to be taken as evidence of such recovery.

These two experiments seem to be the only bilateral operations recorded in the literature. Unilateral lesions were made by Schüller [12] in monkeys and by Ranson [13] in cats. Sagittal sections of the pyramidal decussation, approached from the ventral surface, were made by Rothmann [14] in dogs. This last experimenter, however, found it necessary to use the dorsal approach and to split the entire medulla in the midline in order to section the decussation in monkeys. A similar operation has been used more recently by Langworthy,[15] Pike, Elsberg, McCulloch and Rizzolo [16a] and Pike, Elsberg, McCulloch and Chappell [16b] in cats. Redlich [17] and Probst [18] reported lesions of the pyramid, but gave no details.

Some of the findings in the animals under discussion are of special interest. Thus, the contact placing reactions of Munk and Rademaker

12. Schüller, A.: Experimentelle Pyramidendurchschneidung beim Hunde und Affen, Wien. klin. Wchnschr. **19**:57, 1906.

13. Ranson, S. W.: Rigidity Caused by Pyramidal Lesions in the Cat, J. Comp. Neurol. **55**:91, 1932.

14. Rothmann, M.: Die Zerstörung der Pyramidenbahnen in der Kreuzung, Neurol. Centralbl. **19**:1055, 1900; Ueber experimentelle Läsionen der Medulla oblongata, Verhandl. d. Kong. f. inn. Med. **19**:431, 1901; Ueber die functionelle Bedeutung der Pyramidenbahn, Berl. klin. Wchnschr. **38**:574, 1901; Ueber experimentelle Läsionen des Centralnervensystems am anthropomorphen Affen (Chimpansen), Arch. f. Psychiat. **38**:1020, 1904; Ueber die physiologische Wertung der corticospinalen (Pyramiden-) Bahn, Arch. f. Anat. u. Physiol. (Physiol. Abt.), 1907, p. 217.

15. Langworthy, O. R.: The Area Frontalis of the Cerebral Cortex of the Cat, Its Minute Structure and Physiological Evidence of Its Control of the Postural Reflex, Bull. Johns Hopkins Hosp. **42**:20, 1928.

16. (*a*) Pike, F. H.; Elsberg, C. A.; McCulloch, W. S., and Rizzolo, A.: Some Observations on Experimentally Produced Convulsions. The Localization of the Motor Mechanisms from Which the Typical Clonic Movements of Epilepsy Arise, Am. J. Psychiat. **9**:259 (Sept.) 1929. (*b*) Pike, F. H.; Elsberg, C. A.; McCulloch, W. S., and Chappell, M. N.: The Problems of Localization in Experimentally Induced Convolutions, Arch. Neurol. & Psychiat. **23**:847 (May) 1930.

17. Redlich, E.: Ueber die anatomischen Folgeerscheinungen der motorischen Rindencentren bei der Katze, Neurol. Centralbl. **16**:818, 1897.

18. Probst, M.: Ueber den Hirnmechanismus der Motillität, Jahrb. f. Psychiat. u. Neurol. **20**:181, 1901.

were initially absent but recovered with time to a certain degree. Bard [19] recently reported that this reflex was permanently absent after removal of the motor area in cats. The recovery found in my series therefore indicates that efferent tracts from the motor cortex, other than the pyramidal, are capable of mediating this function to a certain extent. What these other pathways are is not clear from the present experiments, but evidence from other sources suggests the participation of the corticorubrospinal system (Marshall [20]).

The finding of a greater deficiency in the contact as compared with the visual placing reactions argues against the thesis of Lashley [21] that the pyramidal tracts are solely concerned in "mass facilitation," or "priming" the cells of the lower neurons "so that they may be excited by impulses from other sources." To make this hypothesis tenable in the present case, it would have to be advanced that the facilitatory effect can be exercised in different degrees on the same motor mechanism when it is incited through different afferent channels. It must facilitate the tactile more than the visual reflex. To do this it must therefore have connections with both afferent systems, and different amounts of facilitation must be released from stimulation of the two inciting sources. From the afferent to the efferent end, therefore, there must be a specific facilitatory reflex which more or less parallels the main or primary reflex. But this is no different from the orthodox reflex system containing many fibers, any one of which may be conceived as facilitating any other. However, one cannot conclude from these facts that the pyramidal tracts do not mediate a general mass facilitation. It is denied only that this is their sole function.

The defects in the hopping reactions are, as compared with those of Bard's [19] animals which underwent operation on the motor cortex, of less degree.

The finding of increased extensor tone with pyramidal lesions is at variance with the earlier observations of Starlinger,[6] Schüller [12] and Rothmann.[14] More recently, Langworthy,[15] who split the medulla up the midline, found no extensor tonus, and Pike and his co-workers [16] found it only occasionally and confined to the forelimbs. On the other hand, Ranson [13] found it constantly, even in some of his animals with only small lesions in the pyramids. A possible explanation of the report of its absence by the earlier investigators may be that inadequate

19. Bard, P.: Studies on the Cerebral Cortex: I. Localized Control of Placing and Hopping Reactions in the Cat and Their Normal Management by Small Cortical Remnants, Arch. Neurol & Psychiat. **30**:40 (July) 1933.

20. Marshall, C.: On Certain Midbrain Lesions in Cats, Anat. Rec. **58**:26 (March) 1934.

21. Lashley, K. S.: Studies of Cerebral Function in Learning: V. The Retention of Motor Habits After Destruction of the So-Called Motor Areas in Primates, Arch. Neurol. & Psychiat. **12**:250 (Sept.) 1924.

tests were employed to bring it out. In Ranson's experiments and in those of the present series it was most easily observed when the animal was suspended by a ventral sling. Extensor tonus is often decidedly less when the animal is lying in the dorsal position, and it may or may not be noted during standing or walking. Accordingly, if the animal is not tested in a number of positions, an increased resistance to flexion might readily be missed.

On the other hand, the objection might be raised that pressure on the ventral surface of the body, such as occurs in the sling, is a complicating factor, and that the results obtained by this method do not represent the fundamental state of extensor tone. Olmsted and Logan [22] seemed to have had something like this in mind when they reported that in their animals with one motor area removed "flaccid paralysis" developed yet "under appropriate conditions," such as holding the animal in the air in the vertical position or making it sit upright on a table, extensor rigidity could be brought out. The "lying down" position seems to have been regarded by the authors as the fundamental one, and the reactions obtained in it gave the diagnosis of flaccid paralysis.

To me the idea that any one position represents the fundamental state free from complicating influences appears not to be justified. The objection that pressure on the ventral surface probably facilitates and augments the resistance to flexion is at once admitted, with the qualification that it does not produce what is otherwise not there, for the normal controls do not show it. But if ventral stimuli facilitate and augment the response, dorsal stimuli inhibit and conceal it. If one grasps the skin of the cat over the sacrum, the resistance is frequently diminished, as also happens when the animal lies in the dorsal position, a finding entirely in accord with that of Ranson and Hinsey [23] in the *Stütz* reflex in decapitated and spinal cats. Just what position represents the exact mean, free from facilitating and inhibiting influences, it is not possible to say, and it may well be questioned whether such a posture exists. One must form an opinion from an examination of the animal under many conditions, and the possibility of an increased resistance to flexion should not be denied without a comparison of the animal on which operation has been performed and the normal animal in the facilitating positions. In this series, in some postures little or no increase over the normal was found, while in others a definite augmentation was present.

The presence of an increased resistance to flexion from pyramidal lesions is interesting in relation to the observations in animals after

22. Olmsted, J. M. D., and Logan, H. P.: Lesions in the Cerebral Cortex and Extensor Rigidity in Cats, Am. J. Physiol. **72**:570, 1925.

23. Ranson, S. W., and Hinsey, J. C.: Reflexes in the Hindlimbs of Cats After Transection of the Spinal Cord at Various Levels, Am. J. Physiol. **94**:471, 1930.

removal of the motor area. In the monkey, Fulton and Kennard [24] found that spasticity resulted from removal of the premotor area (Brodmann's area 6), but not from that of the motor area itself (Brodmann's area 4). In cats the findings were conflicting: Warner and Olmsted,[25] King [26] and Langworthy [15] reported spasticity after removal of the frontal region, but not after removal of the motor area. On the other hand, Laughton,[27] McKibben,[28] McKibben and Wheelis [29] and Bard [19] stated that spasticity comes from removal of the motor area and not from removal of the premotor area. The present findings could be related to either group, depending on the exact origin of the fibers found in the pyramids. If the fibers of the pyramidal tract take origin exclusively in the motor area, as described in the classic work of Holmes and May,[30] the findings presented here give support to the authors of the second group mentioned. If, however, some fibers arise in the frontal region, these observations do not conflict with the first view. The existence of such fibers, in opposition to the observations of Holmes and May, has been shown by Minkowski [31] in the monkey and by McKibben and Wheelis [29] in the cat. The last mentioned observation, which is most important for this study, is, however, not conclusive, since the authors do not produce sufficient evidence to show that the degenerating fibers in the pyramidal region found after removal of the frontal cortex might not have come from associated damage to the motor area.

The mechanism of the diminished flexor response on passive extension and that of the reduced amount of spontaneous struggling are not clear. Langworthy [15] and Ranson [13] appeared to regard them as secondary to the extensor hypertonus. In the cases under discussion,

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24. Fulton, J. F., and Kennard, M. A.: The Relation of the Motor Areas of the Cortex to Spasticity and Postural Reflexes in Primates, Am. J. Physiol. **105**: 35, 1933; The Localizing Significance of Spasticity, Reflex Grasping, and the Signs of Babinski and Rossolimo, Brain **56**:213, 1933.

25. Warner, W. P., and Olmsted, J. M. D.: The Influence of the Cerebrum and Cerebellum on Extensor Rigidity, Brain **46**:189, 1923.

26. King, W. T.: Observations on the Rôle of the Cerebral Cortex in the Control of the Posture Reflex, Am. J. Physiol. **80**:311 (April) 1927.

27. Laughton, N. B.: Studies on the Occurrence of Extensor Rigidity in Mammals as a Result of Cortical Injury, Am. J. Physiol. **85**:78, 1928.

28. McKibben, P. S.: Experiments on the Motor Cortex of the Cat, Anat. Rec. **42**:57, 1929.

29. McKibben, P. S., and Wheelis, D. R.: Experiments on the Motor Cortex of the Cat, J. Comp. Neurol. **56**:373, 1932.

30. Holmes, G., and May, W. P.: On the Exact Origin of the Pyramidal Tracts in Man and Other Mammals, Brain **32**:1, 1909.

31. Minkowski, M.: Etude sur les connexions anatomiques des circonvolutions rolandiques, pariétales et frontales, Schweiz. Arch. f. Neurol. u. Psychiat. **12**:71, 1923; **14**:255, 1924; **15**:97, 1924.

however, these deficiencies were not directly proportional to the extensor tonus, and they not infrequently persisted after the latter had passed away. Some other factor must therefore be at least partly responsible.

The problem as to whether the failure to correct an abnormal posture imposed on the foot is dependent on injury to the sensory or motor side of a reflex arc or whether it is due to the absence of a general flexor facilitation cannot be answered in the present experiments, because of the associated damage to the medial lemniscus. As to the spontaneous postural defects, it is interesting to note that the grosser disorders, such as crossing of the forelegs, occurred only in the first few postoperative days, when there was a defect in the optic placing reaction as well as in the tactile placing and hopping tests. This agrees with Bard's [19] observations that the tendency of decorticate cats to assume gross abnormal postures is not due to the defects in the tactile placing and hopping reactions alone, but to a visual disorder also.

The reduced activity and deliberative motion seen characteristically in the animals with bilateral lesions and to a lesser extent in those with unilateral lesions are a final problem. Starlinger [6] reported that some of his dogs were slower, more cautious and more deliberate than the normal control, but he attempted no explanation. Rothmann [14] also observed this fact, and stated that the slowness in the movement was the only feature that distinguished monkeys with pyramidal lesions in the later stages from normal monkeys. He explained it on the basis of the reduced total area of the motor fibers from the cortex to the spinal cord. The literature discussing the relations of the phenomenon is conflicting. Langworthy's [15] cats in which the area frontalis had been removed were dull, listless and lethargic, while Olmsted and Logan's [22] were restless and continually on the move. In monkeys, if the area frontalis is removed, there are restlessness and activity (Jacobsen [32]), but if the premotor area is removed with the frontal area, slowness of movement appears (Fulton, Jacobsen and Kennard [33]). The ultimate explanation of these findings is not apparent.

Injury to one pyramid alone produced chiefly contralateral functional defects, but there were some homolateral deficiencies as well. This would appear to be dependent on degeneration of the uncrossed pyramidal tract in the lateral column previously noted. Physiologically, the homolateral hindleg appeared to be more involved than the foreleg. Interesting in this connection are the observations of Bucy and Fulton,[34]

32. Jacobsen, C. F.: A Study of Cerebral Function in Learning: The Frontal Lobes, J. Comp. Neurol. **52**:271, 1931.

33. Fulton, J. F.; Jacobsen, C. F., and Kennard, M. A.: A Note Concerning the Relation of the Frontal Lobes to Posture and Forced Grasping in Monkeys, Brain **55**:524, 1932.

34. Bucy, P. C., and Fulton, J. F.: Ipsilateral Representation in the Motor and Premotor Cortex of Monkeys, Brain **56**:318, 1933.

who have described an "ipsilateral" area in the premotor region of the monkey, which on stimulation gave a response generally seen predominantly in, and in some cases entirely restricted to, the homolateral hindleg.

SUMMARY

1. Following lesions of the pyramids in the medulla (twelve bilateral and five unilateral) the initial symptomatology included the following: (1) diminished or absent contact placing reactions, (2) reduced visual placing reactions, (3) difficulties in walking a ladder or narrow track, (4) delayed and otherwise disordered hopping reactions, (5) increased resistance to passive flexion, (6) diminished resistance to passive extension and a reduced spontaneous struggling in the affected limb, (7) spontaneous postural abnormalities, (8) noncorrection of abnormal postures imposed on the feet, (9) occasional transient staggering, (10) a certain stiffness in the gait, (11) a heavy and awkward landing after jumping down from a height and (12) reduced activity and a slowness and deliberation in motion.

2. In the cases of unilateral lesions the defects were chiefly on the contralateral side. A certain amount of homolateral damage was, however, usually present.

3. In the cases of bilateral and unilateral lesions there were not infrequently differences in the forelimbs and hindlimbs, with a certain tendency for the hindlimbs to appear more disordered. In some cases, however, there was difficulty in equating the tests for the forelimbs and hindlimbs.

4. In all of the symptoms a certain amount of recovery took place. In some it was partial, while in others it appeared complete. There was no constant set of symptoms remaining over at the end of the period of observation of from two to three weeks.

5. Pathologically, the pyramidal tracts were found partly or completely cut. The medial lemniscus was almost always damaged to a certain extent, and the predorsal bundle occasionally showed a small amount of degeneration.

6. The severity and duration of the symptoms generally paralleled the amount of degeneration found histologically. Since the injuries were not confined to the pyramidal tracts, it is probable that pure lesions of their structures would produce a smaller symptomatology than that described.

7. The findings were on the whole of lesser severity than those reported in cases of removal of the motor areas.

Dr. Donald Barron assisted in the development of the operative technic and in many other ways. Mr. G. P. DuShane furnished operative assistance.

Clinical Notes

SERIAL STUDIES OF THE INTELLIGENCE OF PATIENTS WITH EPILEPSY

JOSEPH FETTERMAN, M.D., AND MARGARET R. BARNES, M.A., CLEVELAND

In 1925 we began a program of serial mental tests on patients in the Epileptic Clinic of the Lakeside Hospital Dispensary. We were interested not only in the immediate use of the information but also in its correlation with the clinical progress of the patient.

We present the results in only one of the psychologic tests given, namely, the Stanford revision of the Binet test. In spite of the various criticisms leveled at this procedure, it is perhaps the most valid and reliable single index of abstract intelligence available. The general use of this test and the ease with which the results may be interpreted further recommended it to us. Dawson and Conn [1a] reviewed the important literature and established the reliability of the Binet test for single tests and for retests made from year to year on the same person.

It was originally planned to retest all patients at yearly intervals. However, this program was not achieved in its entirety because either the physical condition of the patient accounted for many broken appointments or the patients dropped from the clinic for short periods or permanently. We tested 105 patients in all, 46 of whom were retested from two to six times at intervals of from one to two years.

In the main, our patients had chronic epilepsy of long standing. The majority had idiopathic epilepsy; in 5 the condition was considered as hystero-epilepsy; in 7 it was due to alcohol, and 18 patients belonged to the group of those with organic epilepsy. All epileptic patients were referred to the psychometrist as a routine, the sole criterion in the selection being the ability to speak English.

The average intelligence quotient for the 105 patients was 74, with a range from 34 to 133 (table 1). The average intelligence quotient for the 14 patients under 16 years of age was 78, while for the adults it was 73. Our averages rate distinctly above those obtained by Fox [2] for the inmates of the Longfield Epileptic Colony. Fox reported an average intelligence quotient of 71 for boys and 65 for girls. Dawson and Conn [1b] determined an average value of 82 for a group of 49 children. Our figures were essentially the same for both sexes.

Read before the Central Neuropsychiatric Association, Cleveland, Oct. 14, 1933.

From the Neuro-Psychiatric Service, Lakeside Hospital, and the Psychological Laboratory, Western Reserve University.

1. Dawson, S., and Conn, J. C. M.: (*a*) Intelligence and Disease, Medical Research Council, Special Report Series, no. 162, London, His Majesty's Stationery Office, 1931; (*b*) The Intelligence of Epileptic Children, Arch. Dis. Childhood **4**:142 (June) 1929.

2. Fox, J. T.: Response of Epileptic Children to Mental and Educational Tests, Brit. J. M. Psychol. **4**:235, 1924.

An attempt was made to separate the 105 cases into subdivisions on the basis of etiology, so as to correlate the intelligence quotient with the type of the disease. This attempt was complicated by the fact that what appeared to be the cause of the epilepsy may have been merely the mode of onset and also by the fact that in several instances a combination of factors was responsible for the attacks. For example, M. M. had her first attack at the age of 26, during a period of great stress and insomnia, when her husband underwent a major operation. The second attack likewise came during a period of emotional strain. As physical examination gave negative results and as the patient was extremely "nervous," we considered her as belonging to the group of patients with hystero-epilepsy. About four years later, headaches and choked disks developed, and the patient was ultimately found to have tumor of the brain.

Without stressing too strongly the boundaries of the subdivisions, our results may be listed as follows: The average intelligence quotient for the hysterical group

TABLE 1.—*Intelligence Quotients of the Entire Group of Epileptic Patients*

I. Q. Level	Number of Patients
30- 34.9	1
35- 39.9	0
40- 44.9	1
45- 49.9	3
50- 54.9	6
55- 59.9	10
60- 64.9	12
65- 69.9	9
70- 74.9	11
75- 79.9	8
80- 84.9	14
85- 89.9	9
90- 94.9	7
95- 99.9	7
100-104.9	4
105-109.9	0
110-114.9	1
115-119.9	0
120-124.9	0
125-129.9	0
130-134.9	1
Total	105

was 81; for the idiopathic group, 77; for the alcoholic group, 70, and for the organic group, 69. The low value for the organic group is due in part to 10 patients with either trauma sustained at birth or infantile encephalitis, most of whom had a distinctly low intelligence quotient.

It was thought that there might be some relation between the mentality of the patient and the length of time he had been subject to attacks. Patients who had had convulsions for five years or less were compared with those who had had the disease for from ten to twenty-five years. The small difference which appeared in the comparison was invalidated by the difference in age in the two groups. Naturally, the patients under 16 were more likely to be included in the group of those who had had the disease for less than five years while most of the adults fell in the second group. It has already been pointed out that the younger patients tended to rate somewhat higher than the adults.

Again, if one compares the patients of lowest, with those of highest, intelligence quotient no uniformity in the duration of the illness is found. The number of major convulsions also appears to be unrelated to the intelligence level. This observation is in line with Dawson's findings.

SERIAL STUDY

Forty-six patients were tested twice or more, the average interval between tests being approximately twenty-one months. Nineteen patients showed a slight increase in the intelligence quotient; 23 showed a moderate loss, and in the remainder the quotient was unchanged. The average of these changes is not larger than the difference which one may obtain between tests and retests on normal persons.

Twenty-five patients were tested three or more times (table 2). The most interesting feature of these serial studies is the fluctuation of the intelligence quotient from one test to another. What may look like a significant change one year may be entirely offset the next year by a change of equal magnitude in

TABLE 2.—*Change in Intelligence Quotient in Three Tests or More*

No.	Patient	Type of Epilepsy	Duration of Epilepsy, Years	Initial I. Q.	Tests					
					1st to 2d	2d to 3d	3d to 4th	4th to 5th	5th to 6th	6th to 7th
1	R. G.	Idiopathic	30	72	— 6	— 2	....	....	...	...
2	B. K.	Alcoholic	5	68	— 3	+ 7	....	....	...	...
3	W. Mc.	Idiopathic	17	62	+ 2	± 0	....	....	...	...
4	J. M.	Idiopathic	10	95	+ 5	—21	....	....	...	...
5	H. O.	Idiopathic	3	83	— 7	+11	....	....	...	...
6	G. T.	Idiopathic	13	69	± 0	+13	....	....	...	...
7	S. B.	Idiopathic	24	99	+ 8	— 6	....	....	...	...
8	B. B.	Organic	10	81	—14	— 8	+ 1	....	...	...
9	L. B.	Idiopathic	7	88	+ 5	— 5	—14	....	...	...
10	L. B.	Organic	7	88	+ 6	— 4	+12	....	...	...
11	M. F.	Idiopathic	15	85	+ 8	— 6	+ 6	....	...	...
12	R. H.	Organic	2	66	— 3	+ 5	— 7	....	...	...
13	E. K.	Idiopathic	14	61	— 1	+ 9	— 4	....	...	...
14	M. K.	Idiopathic	24	76	+ 6	— 4	+ 5	....	...	...
15	L. S.	Idiopathic	13	85	— 2	— 2	+ 5	....	...	...
16	A. C.	Idiopathic	31	89	— 7	+10	— 1	....	...	...
17	L. D.	Idiopathic	15	59	+ 2	+ 4	— 2	....	...	...
18	R. S.	Idiopathic	5	65	— 5	+ 5	+ 1	....	...	...
19	S. B.	Idiopathic	12	64	— 5	+14	— 4	....	...	...
20	M. D.	Organic	7	58	+ 2	— 3	— 3	....	...	...
21	E. H.	Idiopathic	28	60	— 2	— 2	— 3	+12	...	...
22	S. S.	Idiopathic	13	91	+14	— 9	+12	—20	...	...
23	M. H.	Idiopathic	5	86	—17	+ 1	+24	— 8	—2	...
24	M. S.	Idiopathic	19	67	— 2	—15	+14	— 4	—2	...
25	V. V.	Idiopathic	8	73	+ 3	+ 2	— 1	— 2	+7	+4

the opposite direction. Only two patients, J. M. and B. B., showed a decline in the intelligence quotient from both the first to the second test and the second to the third test. B. B. maintained his level from the third to the fourth test. Both patients showed adverse changes in physical condition paralleling these changes in the intelligence quotient. The variations of the other patients appeared to be unrelated to the general condition, although a group of seizures may influence a test made close to the time of attacks.

An instance of such a change is that appearing in the last test made on S. S., which showed a decline of 20 points. This examination was made soon after a series of severe seizures.

For the majority of the 25 patients there was no decided trend toward deterioration in the Binet rating. This result differs sharply from the figures of repeat tests obtained by Dawson and Conn.[1b] These observers found a definite lowering of the intelligence quotient in the second series of tests, from an initial average of 82 to a second average of 66.5.

Our figures lend numerical support to the significant conclusion of Paskind,[3] based on the study of a large group of private (extramural) epileptic patients. Paskind criticized the prevailing concept of mental deterioration in epilepsy, pointing out that this opinion was based on a study of patients in institutions, a group obviously deteriorated. He reported 304 cases in private patients who had had attacks for six years or more, of whom only 20, or 6 per cent, showed deterioration. "The rest carried on in the world the same as their fellowmen." His study challenged the age-old concept of deterioration in epilepsy. However, it must be pointed out that Paskind based his conclusion of nondeterioration merely on the clinical estimate of the patient's ability to continue at his job. Such private patients constitute also an exceptionally select group.

Our ambulatory patients in the clinic were, therefore, appropriate subjects, as they represented a transitional group between the rather intelligent private patients and the deteriorated inmates of institutions.

It is not to be assumed that our results indicate that patients with epilepsy do not show mental deterioration. The majority of our patients had been subject to attacks for a number of years before they were first tested. Deterioration may have occurred before they came under our observation, when it may have reached a constant level or plateau. Again, we may not have followed these patients over a sufficiently long period to detect any slow process of deterioration.

It may be that the change in the epileptic personality cannot be detected by the Binet test. We mention the clinical opinion that the important psychic change in epilepsy is not so much deterioration in intelligence as disturbance in emotion—a change in affect, which one might call a deterioration in spirit. The maladjustment of epileptic patients and the behavior difficulties which they present may arise from frustration, defeat and failure rather than from an inherent cerebral change responsible for a gradually decreasing intelligence quotient. While most persons make progress in their station and position, the epileptic patient, caged by his disease, stands still. By contrast, his relative position becomes inferior. The resulting feeling of futility, and not a change in the intelligence quotient, may lead to the many social difficulties that are common in epilepsy.

Abadie,[4] in a splendid article, selected slowness, or "bradypsyche," as the outstanding characteristic of epilepsy. He laid greater stress on the emotional factors, such as depression, marked changes in mood and irritability. He questioned the accepted view of "an epileptic psychosis." His opinion was that epilepsy does not cause a psychosis, but is merely present along with it. Grossmann,[5] in discussing the character of the epileptic patient, explained the peculiar content of both destructive brutality and religious piety as resulting from "the anxiety of death, reexperienced with each loss of consciousness."

A check on our results is being made at present by administering the Babcock test for mental deterioration at the same time that the Binet test is given. This test is designed to indicate to what extent a patient has deteriorated from his former level.

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3. Paskind, H. A.: Extramural Patients with Epilepsy, with Special Reference to Frequent Absence of Deterioration, Arch. Neurol. & Psychiat. **28**:370 (Aug.) 1932.

4. Abadie, J.: L'épilepsie psychique, Rev. neurol. **1**:1201 (June) 1932.

5. Grossmann, I.: Theorie des epileptischen Charakters, Ztschr. f. d. ges. Neurol. u. Psychiat. **117**:12, 1928.

SEDATIVE THERAPY AND INTELLIGENCE QUOTIENT

Another inference from this serial study is that the intelligence quotient is not materially altered by the continued use of sedative drugs. The majority of the patients included in the study had been taking a sedative, usually phenobarbital, in doses of from 2 to 6 grains daily, over a period of years. As these patients showed no significant deterioration, it is suggested that long-continued sedative therapy per se does not produce reduction in the intelligence quotient.

SUMMARY

We conducted a serial study on the intelligence quotient of patients with chronic epilepsy. The average intelligence quotient for the 105 patients studied was 74. Epilepsy of long duration in which attacks are frequent may be present in a person whose intelligence quotient is normal or above. There was no direct connection between the intelligence quotient and the type, severity or duration of the disease in the subjects studied. The serial study showed substantially no significant deterioration in 46 epileptic patients when they were retested after an interval of one to two years. Sedative medication, even when long continued, did not produce deterioration in the intelligence as measured by the Binet test.

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Dr. C. W. Stone assisted in this study.

A SMALL MENINGIOMA ARISING FROM THE TUBERCULUM SELLAE

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In Cushing and Eisenhardt's [1] article on meningiomas arising from the tuberculum sellae, figure 70 illustrates schematically four stages in the development of these tumors (fig. 1): *I* illustrates a presumptive initial stage. *II* depicts the smallest tumor which the authors had seen, an incidental observation at necropsy in a patient who died from other causes. This tumor was slightly larger than the pituitary body. *III* and *IV* illustrate the larger tumors which present the syndrome of primary optic atrophy, bitemporal field defects and a normal sella turcica.

This article is the report of a case of a very small meningioma, about the size of that shown in Cushing's presumptive initial stage *I*, but flatter and with a larger attachment to the dura over the tuberculum sellae, which was also an incidental observation at postmortem examination.

The patient, a man, aged 47, had suffered for many years from recurrent attacks of nasal sinusitis. Following a flare-up of this condition there developed internal strabismus, visual and auditory hallucinations referred to the right side, a rising temperature, leukocytosis, a stiff neck and a positive Kernig sign. Neurologic examination gave negative results. The spinal fluid was normal. Roentgenograms showed cloudiness of the left maxillary and ethmoid sinuses. The left frontal lobe was explored through a small trephine opening for a possible abscess, with negative results. The patient died two days after operation. Postmortem examination was made ten hours later by Dr. Ernest Hall. The more important anatomic diagnoses were: (1) chronic streptococcic sinusitis, (2) streptococcic septicemia, (3) dural endothelioma and (4) bronchopneumonia. Streptococci were recovered from the lung, spleen, bone marrow and left frontal sinus. Aside from an increased amount of subarachnoid fluid and rather marked congestion of the pial vessels, the brain showed no gross abnormalities on section after hardening or on microscopic examination. When the frontal lobes were lifted from the base of the skull, a small tumor was seen attached to the dura over the tuberculum sellae. This was 9 mm. in diameter, and its greatest height was estimated at 3 mm. The surface was finely granular. The optic chiasm lay between the tumor and the pituitary stalk, touching the posterior margin of the tumor, but it seemed certain that with the normal amount of fluid in the cisterna chiasmatica, the chiasm would not have been in contact with the tumor. On gross examination the tumor had the appearance of a meningioma.

The specimen was sectioned after fixation and decalcification. Figure 2 is a photograph of a sagittal section through the stalk of the hypophysis. It may be seen that in the fixed specimen the anteroposterior diameter of the tumor (8.5 mm.) was slightly greater than that of the hypophysis (8 mm.) and that the vertical diameter of the tumor (2 mm.) was less than half that of the hypophysis (5 mm.). This specimen, therefore, had a more extensive attachment to the dura over the

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From the Department of Surgery, Stanford University School of Medicine.

1. Cushing, Harvey, and Eisenhardt, Louise: Meningiomas Arising from the Tuberculum Sellae, (*a*) Arch. Ophth. **1**:1 (Jan.) 1929; (*b*) ibid. **1**:168 (Feb.) 1929.

tuberculum sellae and considerably less vertical diameter than the presumptive initial stage in Cushing's drawing; it suggests that such tumors start as flattened masses with a wide base, and grow upward to form eventually the more or less

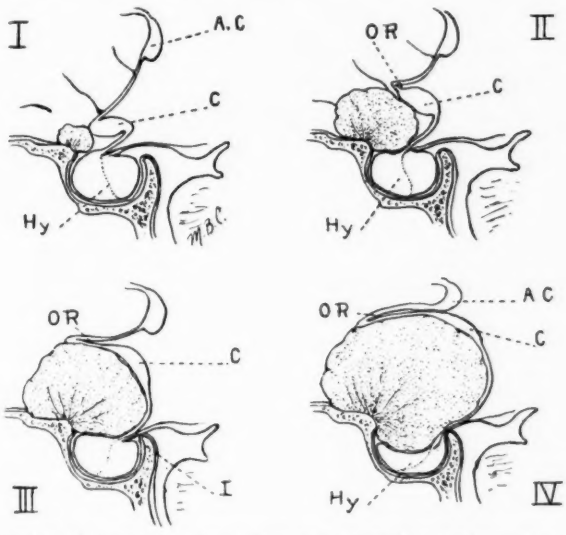

Fig. 1.—Series of drawings illustrating the advancing deformation of the chiasm and third ventricle on sagittal section in four stages; *Hy* indicates the hypophysis; *C*, the chiasm; *O R*, the optic recess; *A C*, the anterior commissure, and *I*, the infundibulum. *I* is a presumptive initial stage; *II* is from a specimen observed at autopsy and is possibly presymptomatic; *III* and *IV* represent the early and late stages producing the syndrome with tumors from about 10 to 18 Gm. in weight. Any growth larger than *IV* is likely to cause hydrocephalus. (From Cushing and Eisenhardt [1b].)

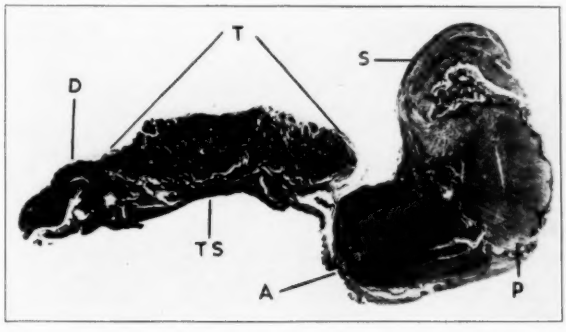

Fig. 2.—Photograph of specimen, × 4. *T* indicates the tumor; *D*, the dura; *T S*, the tuberculum sellae; *A*, the anterior lobe; *P*, the posterior lobe, and *S*, the stalk of the hypophysis.

spherical tumors seen at operation. Microscopically, the tumor was a typical meningioma or dural endothelioma. There was no invasion of the dura between the tumor and the tuberculum sellae or of the bone itself.

SPECIAL ARTICLE

CONTRIBUTIONS OF HASLAM, BAYLE, AND ESMARCH AND JESSEN TO THE HISTORY OF NEUROSYPHILIS

HASLAM'S "OBSERVATIONS ON INSANITY," BAYLE'S "RECHERCHES SUR L'ARACHNITIS CHRONIQUE" AND ESMARCH AND JESSEN'S "SYPHILIS UND GEISTESSTÖRUNG"

MERRILL MOORE, M.D.
AND
H. C. SOLOMON, M.D.
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The literature dealing with the history of dementia paralytica usually credits the first description of the pathology of the disease to John Haslam, the first satisfactory recognition of the disorder to A. L. J. Bayle and the relationship of the disease to syphilis to Esmarch and Jessen. These studies seem to be the three pillars on which the subsequent development of knowledge concerning this disease has been built. It is our purpose to present the material contributed by these authors to the knowledge of the disease and to give a brief critical summary. At the outset it may be stated that the work of Bayle far transcends in importance that of the other two contributors. Indeed, it is doubtful whether Haslam's work would be given much consideration were it not for nationalistic pride.

HASLAM'S CASE FROM HIS "OBSERVATIONS ON INSANITY"

John Haslam's contribution to the subject is contained in his "Observations on Insanity: with Practical Remarks on the Disease, and an Account of the Morbid Appearances on Dissection." This work was published in London in 1798 by Haslam, who signed himself as "Apothecary to Bethlem-Hospital," the title of "Apothecary" corresponding to what would now be called "resident physician." His work consists of a brief report of twenty-nine consecutive cases which came to autopsy with a brief clinical description and even briefer summary

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of the pathologic changes. In addition there is a rather short discussion of the general subject of mental disorder and its causes. In his description of cases, Haslam did not endeavor to classify the cases or draw significant conclusions other than to make the following statement: "The causes which I have been enabled most certainly to ascertain may

OBSERVATIONS

ON

INSANITY:

WITH

PRACTICAL REMARKS ON THE DISEASE,

AND AN

ACCOUNT OF THE MORBID APPEARANCES
ON DISSECTION.

By JOHN HASLAM,

LATE OF PEMBROKE-HALL, CAMBRIDGE,
MEMBER OF THE CORPORATION OF SURGEONS,
AND APOTHECARY TO BETHLEM-HOSPITAL.

" Of the uncertainties of our preſent ſtate the moſt dreadful and alarming is the uncertain continuance of reaſon."

Dr. JOHNSON's Raſſelas.

London:

PRINTED FOR F. AND C. RIVINGTON, NO. 62,
ST. PAUL'S CHURCH-YARD;
AND SOLD BY J. HATCHARD, NO. 173, PICCADILLY.

1798.

Fig. 1.—Facsimile of the title page of Haslam's "Observations on Insanity."

be divided into physical and moral." In fact, he went so far as to say: "It may be a matter for much diversity of opinion whether these morbid appearances of the brain be the cause or the effect of madness; it may be observed that they have been found in all states of the disease." However, he did offer the decided opinion that diseases of the mind must be associated with disease of the brain.

Of the twenty-nine cases described by Haslam, four may conceivably have been cases of dementia paralytica. The credit for having described this disease, however, rests chiefly on one case of the series, case 15, which we quote here in full.

Case XV. J. A., a man forty-two years of age, was first admitted into the house on June 27, 1795. His disease came on suddenly whilst he was working in a garden, on a very hot day, without any covering to his head. He had some years before travelled with a gentleman over a great part of Europe: his ideas ran particularly on what he had seen abroad; sometimes he conceived himself the king of Denmark, at other times the king of France. Although naturally dull and wanting common education, he professed himself a master of all the dead and living languages; but his most intimate acquaintance was with the old French; and he was persuaded he had some faint recollection of coming over to this country with William the Conqueror. His temper was very irritable, and he was disposed to quarrel with everybody about him. After he had continued ten months in the hospital, he became tranquil, relinquished his absurdities, and was discharged well in June 1796. He went into the country with his wife to settle some domestic affairs, and in about six weeks afterwards relapsed. He was readmitted into the hospital August 13.

He now evidently had a paralytic affection; his speech was inarticulate, and his mouth drawn aside. He shortly became stupid, his legs swelled and afterwards ulcerated: at length his appetite failed him; he became emaciated, and died December 27th, of the same year. The head was opened twenty hours after death. There was a greater quantity of water between the different membranes of the brain than has ever occurred to me. The tunica arachnoidea was generally opake and very much thickened; the pia mater was loaded with blood, and the veins of that membrane were particularly enlarged. On the fore-part of the right hemisphere of the brain, when stripped of its membranes, there was a blotch, of a brown colour, several shades darker than the rest of the cortical substance. The ventricles were much enlarged, and contained, by estimation, at least six ounces of water. The veins in these cavities were particularly turgid. The consistence of the brain was firmer than usual.

From reading this record one can conclude that there is described a grandiose type of dementia paralytica in which the patient, after the onset of the disease, went into a state of remission, after six weeks relapsed and then showed the cardinal symptoms of paralysis, speech defect and mental deterioration. On the pathologic side, Haslam described an increase in the subarachnoid fluid, thickening of the arachnoid membrane and internal hydrocephalus, and increased consistency of the brain, all of which are characteristics of the pathologic changes seen in dementia paralytica as it is now understood.

Robertson,[1] who made an extensive study of the matter, stated that this is the most important contribution to the study of dementia paralytica that Haslam produced. Three other cases of the series, cases 10, 16

1. Robertson, G. M.: The Discovery of General Paralysis, Brit. M. J. **2**: 1120, 1922; J. Ment. Sc. **69**:1, 1923.

and 28, may have been cases of dementia paralytica, but it is doubtful whether the thesis that they are could be maintained from the material given by Haslam. It is fair to conclude, therefore, that while Haslam unquestionably described in his report a case of dementia paralytica, its significance was lost on him and was not at that time apparent to the medical profession. In fact, after Bayle's work had been produced, a considerable time elapsed before a retrospective analysis of the literature led to the discovery of this description.

THE THESIS OF A. L. J. BAYLE (1799-1858)

Bayle's contribution to this subject appeared in 1822, when he was about 24 years of age and was published as his thesis for a doctorate of medicine. It appeared under the title, "Recherches sur l'arachnitis chronique, la gastrite et la gastro-entérite chroniques, et la goutte, considérées comme causes de l'aliénation mentale; par A. L. J. Bayle." [2] The portion of the work entitled "Recherches sur l'arachnitis chronique" deals with the subject at hand; it is given herewith in as good an English translation as we have been able to make. The thesis is based on a careful analysis of the clinical and pathologic observations in six cases. A perusal of Bayle's résumé shows clearly that he recognized a disease entity characterized by a clinical syndrome, an invariable course and specific pathologic changes. That his concept of the disorder was extremely broad and consistent with present-day ideas is borne out by the statement, "its gravity and its final results, nearly always tragic, and its wide social complications, deserve to have fixed on it the attention of the entire medical profession." The résumé is so concise that there can be no reason for discussing or analyzing his statements. Unquestionably, on the basis of this work Bayle justified the acceptance of a mental disorder or rather a characteristic symptomatology based on a well defined pathologic condition of the brain and its membranes.

THE VALUE OF BAYLE'S WORK

Bayle's contribution met with the greatest success that can come to any medical contribution in that it led to widespread consideration and discussion. In fact, the author was honored by many severe criticisms, not to mention personal attacks. As a result, the question of chronic arachnitis became a lively issue in the medical world, and from this original description of six cases and the conclusions drawn the subject has grown by addition to the present state of knowledge concerning dementia paralytica.

Bayle is to be accredited not only with having set aside a definite disease entity from among the group of mental disorders, but also

2. The book was published in Paris by Didot LeJeune in 1822.

with having laid the basis for an understanding of the part which pathologic changes in the brain play in the development of mental disorder. In this regard he went further than Haslam and may be considered as the father of the concept that a specific disease of the brain may be the cause of a specific mental disease.

RECHERCHES

SUR

L'ARACHNITIS CHRONIQUE,

LA GASTRITE ET LA GASTRO-ENTÉRITE CHRONIQUES,

ET LA GOUTTE,

CONSIDÉRÉES COMME CAUSES DE L'ALIÉNATION MENTALE;

PAR A. L. J. BAYLE,

DOCTEUR EN MÉDECINE de la Faculté de Paris; Bachelier ès-lettres; ancien Interne de deuxième classe des hôpitaux et hospices civils de Paris; Interne en médecine à la maison royale de Charenton.

Oft from the body, by long ails mistun'd,
These evils sprung, the most important health,
That of the mind, destroy; and when the mind
They first invade, the conscious body soon,
In sympathetic languishement declines.
ARMSTRONG, *Art of preserving health*, b. IV, 134.

A PARIS,

DE L'IMPRIMERIE DE DIDOT LE JEUNE.

Imprimeur de la Faculté de Médecine, rue des Macons-Sorbonne, n.° 13

1822.

Fig. 2.—Facsimile of the title page of Bayle's thesis.

TRANSLATION OF BAYLE'S THESIS: "RESEARCHES ON MENTAL DISEASE"

Preliminary Considerations.—If there is one branch of human diseases that is hardly apt to be carried to a higher point of perfection regarding the description of symptoms, that branch, without doubt, is that of mental diseases.

The famous professor, Pinel, in his "Treatise on Mental Diseases," and, after him, Doctor Esquirol, in his excellent articles in the "Dictionary of Medical

Sciences," have traced out tables of the numerous disorders of the intelligence that are so true to life and so complete that one may search in vain for any symptom or phenomenon of importance that may have escaped these observers. Unfortunately for the human race, the efforts of the greatest medical scientists have been able to remove only a part of the veil that covers the subject with which we are concerned. The symptoms of mental disturbance with its many variations, the causes that prepare the way for it or that excite it have been described with the greatest exactness, but its intimate nature or certainly its immediate causes have escaped to this day the most laborious and the most scholarly studies, and in most instances only more or less hazardous hypotheses have been produced.

Therefore, it is this part of the pathology of mental diseases to which the attention of all physicians should now be attracted and which should invoke the most assiduous observation. Knowledge about the nature of diseases is the most unassailable basis for therapy, and no one doubts that if the nature of mental disturbance were uncovered the treatment of this condition would be so limited, as is the case now-a-days, to the aid of hygienic measures that are often fruitless.

The opinions of authorities relative to the cause and nature of mental disturbance are extremely various. Many distinguished physicians, guided by severe judgment and fearful of falling in error, limit themselves merely to observing, as profoundly as they can, and give no advice and express no opinion on the nature of mental disease.

Pinel set aside no special article in his immortal work for this interesting subject; however, in one place (page 141) he insinuated that the primary seat of mania is in the region of the stomach and intestines, whence spreads, by a sort of radiation, the intellectual disorder or disturbance. Other famous observers such as Bonet, Morgagni, Meckel, Greding and Willis, have put forward different ideas about mental disease, but they are founded on too small a number of facts to deserve much confidence. Most modern physicians regard mental disturbance as the result of irritation of the brain and not as a lesion of the intellectual principle itself. But what is the nature of this physical alteration? Here, precisely, is the obscure and uncertain point. On one hand, numerous anatomic researches made on the bodies of insane people have proved in an incontestable manner that in the majority of these cases there is not any appreciable organic lesion, so far as can be observed, either in the brain and its parts or in the structures dependent to it. Yet, on the other hand, it is known that under other circumstances there do exist more or less striking changes in different organs of the body systems. These facts have served as the basis of opposing opinions about the seat of mental disease and have brought some to look on mental disease as an idiopathic disorder of the brain and others to regard mental disease as symptomatic of disease of this organ.

Georget, the author of a new treatise on insanity, maintains that this disease is always an idiopathic cerebral disorder, that the cause of it is unknown and that the symptoms of it that affect the different organs of the body more or less remote from the brain are secondary or sympathetic to the alterations in the brain. Falret, in his "Treatise on Suicide and Melancholia," holds the same opinion. On the other side, Prost ("A Glance over Madness") regards mental disturbance as an illness that is always sympathetic and defines it as: "a trouble with the cerebral organs that is determined by trouble in the mucous organs in the abdomen, especially the bile and the stomach." According to this author,

most of the predisposing and occasioning causes of mental disease act on the liver and the intestines and through the intermediary of the brain. Thereupon results an increasedly abundant secretion of bile, which flows out into the intestines and there acquires, he claims, properties that seem to be toxic for the brain and corrosive for the digestive apparatus. According to Prost the accumulation of this bile becomes the most active cause of insanity, as it *communicates to the blood certain perverted fluids* and acts in an immoderate way on the mucous membrane of the intestine, which membrane it may inflame or even excoriate, and finally it irritates the intestinal worms which, according to this author, are so often found in cases of mental illness. It is because of these ideas that emetics, purgatives and anthelmintic remedies are used as the basis of treatment for mental disease. Broussais believes that mental disturbance is most frequently accompanied by and dependent on chronic gastritis.

All these opinions about the cause and nature of mental disease appear to be badly founded, merely in that they are too exclusive. It seems to us that every physician who is not dominated by any preconceived idea and will study carefully the phenomena of mental disease will be able to make out some causes and to compare the symptoms of the disease with the anatomic lesions which he may find on opening the bodies of patients who have died. He will not be able to deny that mental disease is most often idiopathic, but sometimes he will find it symptomatic. This is the opinion of Professor Royer-Collard, whose opinion carries great weight in this important subject, and this opinion is the one that we are guided by and we shall try to support and prove by a certain number of facts, and we shall limit ourselves to reporting the cases that tend to show that mental disease may be symptomatic, because the cases that show that mental disease is idiopathic are so numerous and so inclusive that it would be superfluous to make any additions to them.

Our work will be divided into three parts. In the first we shall try to prove that mental disturbance is sometimes the symptom of a chronic inflammation of the arachnoid. The second will have as its object to show that mental disturbance can be caused, maintained or modified by a chronic gastritis or gastroenteritis. The third will include two observations in which madness seemed to have been determined by an atypical gout.[3]

The observations that make up the subject of this dissertation have been made and collected at the Charenton Royal Hospital, under the supervision of Professor Royer-Collard, physician-in-chief of this institution, to whom I wish again to acknowledge here my gratitude for the kindness he has never ceased to show me in the course of my medical studies.

I have performed most of the necropsies conjointly with my friend Dr. Roberts-Roche of the medical service of Charenton Hospital.

OBSERVATIONS ON CHRONIC ARACHNITIS WITH MENTAL DISTURBANCE.

FIRST OBSERVATION: CHRONIC ARACHNITIS, MONOMANIA, DEMENTIA

Claude-Francois L., of a quite robust general make-up, 48 years old, was a lemonade seller who had drunk excessively in the past, who had indulged immoderately in the pleasures of venery, and who had recently met with losses that had caused him great sorrow.

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3. Parts two and three of this thesis are omitted from this translation.

In April 1818, he suddenly lost consciousness, which he regained a short time afterward. This attack was followed by a hemiplegia of the right side, which slowly disappeared; from then on his intellectual faculties were disordered and there followed a sort of "ambitious *délire*" [4] which dominated the patient incessantly, and which gradually increased.

In the month of October he is the Emperor Napoleon, he has immense wealth; forty thousand barrels of gold; but on other subjects his ideas keep a certain coherence. Ordinarily calm, he is sometimes extremely excited, and even violent if he is opposed. He speaks with great difficulty and walks with a staggering gait. He does not sleep; he eats well, and his legs are slightly edematous.

On October 27 he is brought into Charenton Royal Hospital in a demented state, with ideas that are predominantly ambitious, and with his partial paralysis more advanced.

During the first two months little change is noted in his condition.

On Jan. 4, 1819, there are general paleness and flaccidity; sensations are obtuse, and no attention is paid to objects around him, nor does he busy himself with things near him. He hears with difficulty any questions put to him, and in order to make him understand anything it must be repeated to him several times and presented to him in several different manners. He retains only a few incoherent ambitious ideas, which he expresses when he is spoken to. No matter what he is asked his replies are that he is emperor, that his two sons are emperors, that they live at the Tuileries, that he has millions and still more millions and that his wife has three *croix d'honneur*. On any other matter he is unable to associate even the simplest ideas. If he is asked of what country he is emperor, he replies "of Besançon," to which he joins Germany and France.

He can hardly stand up; he walks slowly, staggering with each step he makes, and dragging his feet. He is calm, apathetic and silent, and stays usually in an armchair to which he is tied to keep from falling. At other times he walks in the yard or halls. At moments he talks in a low voice, repeating endlessly the words *emperor, millions, diamonds,* etc. His voice is broken and trembling; his pronunciation is very difficult. He is incontinent of feces and urine. He eats well and asks for food often, but he is very thin.

On January 14, he cannot get up; his face is greatly altered; his faculties are more obliterated, and he has excoriations on various parts of his body.

On January 30, there are weakness, emaciation, edematous legs, continual silence, no reply to questions put to him, except occasionally when they relate to his "ambitious *délire*," in which case he says merely, "emperor, I am emperor . . ."

On February 8, there is complete physical and mental prostration. He cannot offer a single word or make any motion to indicate that he understands questions. His face is disturbed by slight convulsive movements, and his eyes are fixed and inexpressive. All the parts of his body that are exposed to pressure or even slight rubbing, such as the sacrum, the trochanters and the elbows, have become the site of livid excoriations and gangrenous sores. The pulse is weak and slow, and these symptoms increase. On February 9 there is a period of agony lasting a few hours, then death.

Opening the Cadaver.—The body was extremely thin; the legs and feet were infiltrated, and there were scabs on various parts of the body.

4. As we have been unable to find an exact equivalent in English, the French word *délire* has been retained. It designated a delusional state, rather than "delirium."

Skull: A slight serosity exists between the two layers of the arachnoid; from 4 to 6 ounces of the same liquid escape at the base of the skull. The "arachnoid membrane of the dura mater" is greatly injected. The two cerebral hemispheres are adherent in the great fissure. The cerebral arachnoid at the base of the brain is healthy; that of the convexity and of the inner surface of the hemispheres is opaque, whitish in some places and in other places keeping part of its transparency, thickened, very firm, and (on this account) easy to remove from the surface of the cerebrum, to which it is also adherent in several rather extensive regions. The pia mater is very red, injected, and infiltrated, with a great quantity of serous fluid. The lateral and medial ventricles are distended by this same fluid; the membrane that lines these [ventricles] as well as the ventricle of the cerebellum is greatly thickened, can be separated from the cerebral surface and is studded with granulations that one can feel when one touches them, which have made this lining rugous, uneven, wrinkled and somewhat like the surface of shagreen leather. The cerebral substance is soft; the medullary substance is much more firm.

Thorax: All the pectoral organs are healthy.

Abdominal Cavity: The gastric mucous membrane is thickened, rough and "crying under the instrument." Other organs are healthy.

Does not this observation prove that the disorder of intellectual faculties that occurred in the case of L. was the symptom of a chronic arachnitis and not an essential *délire?* It is a conclusion one cannot refuse to admit, when comparing the organic lesions with the symptoms the patient has presented during the course of his illness. It is not necessary to prove that the opacity of the arachnoid, its thickening and the increase of its resistance in the superior region of the cerebrum, as well as the serous infiltration of the pia mater and the discharging of a great quantity of serosity, whether it be at the base of the cerebrum or in its cavities, are the anatomic characteristics of chronic mental disturbance. This is a fact that no one will deny, and all that we know about chronic inflammation of serous membranes, particularly the arachnoid, puts it beyond doubt.

If that is agreed on, let us see what must have been the effect of organic changes in this last-mentioned membrane in determining the phenomena of the illness.

1. At the start, sudden loss of consciousness, hemiplegia; a few instants after, return of feeling and motion of the paralyzed side, but motor embarrassment noted throughout the muscular system; and from this moment on, trouble with his intellectual faculties, monomania. A part of these symptoms belong to apoplexy, but the absence of extravasation of blood, or of a cavity or cyst in the ventricles or the substance of the cerebrum, shows that there had not been a cerebral hemorrhage. The attack that the patient had then must have been what M. Rochoux ("Researches on Apoplexy") calls a "blood-shock" or "coup de sang," that is to say, a sudden "catching" of this fluid in the vessels of the pia mater and the cerebrum; but in this case there is a symptom that one does not see in simple cerebral congestions, that is, *délire*. I attribute this symptom to the irritation of the arachnoid, which, secondarily, irritates the brain; for if it does not exist in cerebral congestion, this affection is not at all accompanied by any modification in the arachnoid. Thus, this first period of the illness presents, on one hand, a blood congestion in the tissue of the pia mater and of the cerebrum, plus a slight irritation of the arachnoid and, on the other hand, the symptoms of each one of these affections.

2. In the second period, difficulty of speech, staggering gait, agitation from time to time and sometimes violence, faculties greatly impaired, with the same ideas dominating. If one recalls that, in cases of "coup de sang" or blood-shock, the paralysis becomes progressively less instead of increasing, when it is correctly treated, one will attribute these phenomena to the increased inflammation of the arachnoid and the serous outpouring (exhalation), changes which work in two ways against the brain; first by irritating it, and second by compressing it.

3. In the last period, progression, trembling, loss of sphincter control and complete dementia with weakening of intelligence, which is reduced to a few dominant and incoherent ideas; no agitation. Then occurs, at this epoch of the disease, what takes place in most of the chronic inflammations of the serous membranes. The slight irritation with which the arachnitis was accompanied disappears. The tissues of the cerebral serous membrane remain opaque and thickened. A great quantity of serous fluid is emitted; the brain is no longer excited, but it is weakened and depressed by the abundant serous fluid into which it is immersed. The following observations will confirm these ideas, which one might regard as too hazardous in this place.

SECOND OBSERVATION: CHRONIC ARACHNITIS. MONOMANIA, MANIA

M. Edme-Charles A., 45 years old, of full-blooded temperament and robust constitution, clerk for a justice of the peace, had undergone much strain and privation on a voyage to the Great Indies; he had indulged in excessive venery and had had several syphilitic diseases. In March, 1817, he had a hemorrhagic dysentery which lasted a good while, then stopped and did not recur. About the same time he was prey to very great worries over the fate of members of his large family, whom he feared he could not support because of the hardness of the times.

About the first of May, he suffered frequent lapses, and was taken, shortly afterward, with an "ambitious *délire*" characterized by ideas of richness, grandeur, honors and by a cerebral congestion marked by difficulty in pronunciation and in walking. He manifested a strong penchant for drinking and the pleasures of love and was subject to exalted moods, and when he was in these he spoke volubly about the objects of his *délire*. He slept very little and ate a lot. (He was bled from the arm with two palets).

On June 27, he was brought to the Royal Institution of Charenton, with ambitious ideas predominant. He is extremely rich and powerful and in a considerably elated state. He is furious at moments in which he tears off his clothes as unworthy of a man of his rank, has difficulty in pronouncing certain words, has a very self-assured manner of walking, is incoherent in his remarks, is voraciously hungry, steals whatever he passes near, partly to satisfy his hunger and partly because everything in the world belongs to him. During the month of July he remained in the same condition.

In the three months that followed, his intellectual faculties became more profoundly altered. He responded sometimes to questions put to him after they were repeated several times, but always in monosyllables or by words entirely irrelevant to the question. His memory was obliterated, and his ideas were very narrow, limited to matters of wealth, grandeur, etc., which came to his mind without connection and without order. He showed no trace of judgment, was calm at some moments and agitated at others (a strait-jacket had to be used to keep him from tearing off his clothes). Speech was drawled, impeded and tremulous; gait extremely staggering and slovenly. Toward the end of Novem-

ber, there is marked amelioration of symptoms with diminution of agitation, with incomplete paralysis and with intelligence much less disturbed.

In December, there is a return of reason, but in weak spirits with no marked *délire*. A few grandiose notions persist that he coordinates fairly well. He wishes first to study pharmacy, next to study medicine "to support his babies." He replies always that he feels "perfectly fine," and is very much satisfied with himself; he wants to get back to his family and take up his job again, if he can. There are slight dysarthria, which causes a certain slowness in his speech and slight faltering over certain words, and a little tremor of the hands. In January 1818, there is diminution of these symptoms. The same condition exists till the end of May.

In the first days of this month he talks more than usual, shows elation in his ideas and behavior, is very self-satisfied; his face is red and animated, and he produces some extravagant propositions for which he must needs be locked up again.

A little later, there are complete upset of the faculties of judgment, a general *délire* accompanied by continual agitation and extreme violence. It is necessary to use the "camisole" (strait-jacket) and the "panier" [5] to restrain him. He does not stop one minute, night or day, shouting confusedly and uttering inarticulate, incoherent cries and vociferations. His voice is thick; no one can understand the words the way he pronounces them, and his face is wan and distorted with very much disordered movements. He makes a thousand efforts to escape; he turns and twists ceaselessly in every direction. His extremities and head undergo spasmodic movements which tax the strong ties that are used for restraining him. In spite of all these disturbances in related functions, his appetite is voracious; his pulse and other nutritional functions continue without alteration.

He continued in this state for about four months, at a sustained level, with slight remissions of brief duration. Toward the middle of November the agitation diminished; his excretory functions became *involuntary;* his legs became edematous, and his mental faculties were effaced. On December 6, he recognized nobody; his respiration grew deep; the pulse grew weak, and he died at 2 o'clock in the afternoon.

Opening the Cadaver.—Skull: Six ounces (about) of serous fluid were drained from the base of the cranium, and much of this flowed out of the rachidian canal. The cerebral arachnoid, over all the surface of the hemispheres, but principally toward the middle part of their external surface, was opaque, grayish, greatly thickened and firm, in such a way that one could easily tear it off from the brain without breaking it. The cerebral arachnoid was intimately adherent to itself in the great fissure, and the inner surface of the arachnoid was stuck to the cerebral parenchyma in the thickened places, so that it was covered with a light soft layer of the latter when one took it off the brain.

The external surface, somewhat injected, presented several rather thin granulations that could be seen but not felt. The arachnoid layer of the dura mater showed a slight redness in different places. The pia mater was red and slightly infiltrated with serous fluid. The lateral ventricles were full of the same fluid;

5. This is a kind of casket, as long as a man's body, made of willow, like ordinary baskets, having a cover, one end of which is left open to permit the head to remain free.

their surface was covered with granulations. The entire cerebral vascular system was enormously injected. The cerebral substance was healthy.

Thorax: Half a pint of serous fluid was found in each pectoral cavity. The left lung was adherent to the costal pleura. Three ounces of serous fluid were found in the pericardial sac. The tissues of the membrane enfolding the heart were greatly infiltrated with this fluid and had a gelatinous appearance.

Abdominal Cavity: There was a small amount of serous fluid in the peritoneum. The gastric mucosa showed a reddish-brown color and was rather thickened and covered with a cast of thick mucus. The capsule of the spleen, adherent to the diaphragm, was hard, whitish, and creaked under the knife, presenting a cartilaginous structure. The other organs were healthy.

The story of the illness just described indicates a striking relationship between symptoms and the observations on the body at autopsy, in which instance, when the latter are given, we can come to some sort of understanding about the former.

To make more strongly felt the coordination and the intimate linking between these causes and these effects, we may place at the head of the following discussion this concise table of organic lesions: an arachnoid membrane that was opaque, grayish, greatly thickened, very tough, adherent to the gray matter of the brain over a great part of the convexity and of the internal surface of the hemispheres, six ounces of serous fluid at the base of the skull and ventricles full of the same fluid with their linings showing marked granulations.

What have been the actions of these alterations in producing the physical and intellectual disorders that have come to our attention in this case? At the start of the disease we have a man of full-blooded temperament. After excesses of all kinds there was a bloody flux, at first habitual, which then stopped; then severe worries that brought about a cerebral congestion. This showed itself in a disturbance of pronunciation and difficulty in walking. At the same time the arachnoid became irritated, and this brought on a monomanic *délire* and agitation that accompanied it from time to time. This last factor, which we have already spoken of, is of the highest importance and needs a further consideration to put its existence beyond question or doubt. The examples of sudden congestion of blood without cerebral hemorrhage are very numerous and make up the largest number of apoplexy cases that one sees every day. The symptoms of this malady are limited to a more or less profound alteration in feeling and movement, which never goes so far as absolute anesthesia or complete paralysis of one side of the body; these symptoms go away more or less promptly, and during their course the patient is not disturbed by *délire* or agitation. On the other hand, the numerous observations that Parent and Martinet have published, and those which one reads about in different dissertations, show us constantly these two phenomena, but in infinitely varying degrees according to a large number of circumstances. One must, therefore, in the observations which we examine attribute the intellectual disturbance and the disorder of movements to the inflammation of the arachnoid membranes. But this inflammation never has the idiosyncrasies of acute insanity; quite light in all the stages of its course, it increases gradually; it changes in a slow but progressive way the tissues of the arachnoid. It shows throughout all its course a chronic progression and is determined by causes that are peculiar to it. It never follows acute arachnitis. It is this last circumstance that caused the last two authors cited to claim that there is no such thing as chronic arachnitis or at least that it must be excessively rare. For the same reason Dr. Montfalcon doubts its existence ("Dictionnaire des sciences médicales," article "phrénésie").

After the first stages of Mr. A.'s disease the inflammation of the arachnoid increased; the serous fluid was emitted more abundantly than in the natural state, and the brain was more irritated and more compressed than it was before this attack; thus came on the motor difficulty, the slight and transient elation and the limited and exclusive *délire* that were observed at this period, followed by maniacal agitation in which the patient had rages and frequent paroxysms of furor, general disturbance of judgment and general incomplete paralysis that rendered pronunciation very difficult and that made the gait staggering. Five or six months afterward, the inflammation subsided and a part of the serous fluid was probably absorbed; then calmness and reason were gradually reestablished in the patient, but his mental faculties remained weak, his motions only recovered a portion of their former freedom, which are phenomena related without doubt to a certain thickening of the arachnoid and to the definite quantity of serous fluid which survived the inflammatory irritation and the blood congestion by which it is always accompanied. Thus, one can conceive how in such a condition the patient must be disposed to have a relapse, even from the slightest of causes. This relapse did take place; a new cerebral congestion manifested itself, and the arachnoid became inflamed again. Then this membrane, irritated more extremely than it had ever been before, thickened more than ever and acquired points of adherence to the exterior surface of the brain over all the spread of its superior surface, and conjointly with a pia mater that was markedly injected. From that necessarily resulted the most active irritation of the brain and, as would necessarily follow, a corresponding disorder of the functions of the brain and the motor apparatus over which it presides, an extreme disorder manifested by complete upsetting of judgment and reason, confused crying out, inarticulate, continual yelling, and ceaseless agitation and violent and spasmodic movements of the head and extremities. These symptoms differ from those accompanying acute arachnitis only as regards their duration. In this last disease they are soon followed by a state of collapse, whereas in the patient who is the object of this discussion they lasted throughout four months.

The period of prostration finally came for Mr. A. one month before his death. A calmness came over him; his intelligence remained obliterated, and his paralysis was marked. With these symptoms one recognizes a more abundant emission of serous fluid. In the observation on L., in which we did not find that the meninges had adhered to the brain, the agitation existed only in the second period of the disease and was not continual or very intense. What influence did the chronic gastritis that was found at autopsy have on the mental disturbance with which this patient was stricken? It is hard to answer this question. But one can be sure that it did not cause the *délire* since there was no symptomatology referable to the stomach when the *délire* manifested itself, and it was only after his being stricken that the patient surrendered himself passionately to drinking.

THIRD OBSERVATION: CHRONIC ARACHNITIS. MONOMANIA, DEMENTIA, IDIOCY

M. Jean-Francois M., of full-blooded temperament, 41 years old, a skilful sculptor, was subject to attacks of abundant and periodic bloody flux that, since the application of leeches, had been stopped for some time and had not reappeared. This man had had several falls from heights of 40 or 50 feet, in which he had suffered a violent cerebral concussion and a fracture of the right arm. In the first few days of January 1818, he underwent a remarkable change in personality, in which his character and his spirit altered greatly and he began to

talk a great deal. He became elated and went into a rage whenever he was crossed in the slightest way. He wanted to transact business with everybody. He showed in his domestic affairs a marked weakening of memory. From then on his intellectual faculties grew deranged in a rapid manner, and some time later his elation diminished. There followed an exclusive and ambitious *délire;* he had fifteen thousand pounds of income; the gigantic projects he was going to carry out were going to bring him millions. He spoke with a little difficulty, and his gait was unsteady. He did not sleep at night and was often drowsy during the day. On Aug. 26, 1818, he came into Charenton, in just about this state. He was able to work, asked for his instruments and drew all over the walls of his room designs that were remarkable for the exactness of their proportions. However, his mind weakened each day; his movements became more difficult. His "ambitious *délire*" grew more and more expansive; he possessed six hundred thousand francs; he was going to build a château finer than the Tuileries and the Louvre. It became impossible for him to do anything in a consecutive way.

On Jan. 14, 1819, his attention could not be gained; there was slight deafness; conception was slow for even the simplest questions; his remarks were vague, and his *délire* was more extensive. He has six billions of money, two hundred ships; he has just bought Italy and is considering taking possession of Asia. He is the best artist in Europe. When asked about his health he replies that he is fine, that he does not need ever to be locked up, that his carriage is at the door and that they are waiting for him there. He still shows considerable good taste in sculpture and design, although he is incapable of doing any reasoning. A few days later his faculties are much feebler; his ideas are incoherent, and his memory is so altered that he cannot remember the names of his nearest relatives and it is only with difficulty that he recalls his wife's name. He is elated and disturbed at moments and his reactions are exaggerated by the slightest opposition. His voice is slow and impeded; his words are spoken falteringly; he walks with difficulty, staggering at times, and at times shows slight tremors of his arms; his appetite is voracious, and there has developed much obesity.

On January 31, his irritability was increased. The dominant ideas were more expansive and incoherent; his gait was so staggering that he walked often like a drunken man.

On February 8, his faculties were profoundly altered. He understood none of the questions put to him and answered with extravagant words which had no connection with what had been asked. The dominant ideas that he had were of wealth, grandeur and power, though there was no kind of coherence between these ideas. He had destroyed all of Asia, of which he was the Emperor; he had broken a bridge that went to the moon; he led the Chinese to Paris, and he was 800 feet high.

On March 15, he was in a state of complete dementia, with the same ideas dominating that made up the whole sphere of his thoughts. In walking he dragged his feet slowly and was always about to fall; his legs could barely support the weight of his trunk, and his tongue was so clumsy that he spoke with extreme difficulty. His urine and feces were no longer under voluntary control; he had a voracious appetite and was generally weak. This patient stayed about six months in this state without showing much change.

On August 23, he had physically improved; his incomplete paralysis had diminished; he spoke with less difficulty and resumed voluntary control of the urine and feces. He appeared demented and somewhat agitated, though not

violent, with frequent outbursts of short duration, leaving no particular impression; his face was stupid, and he looked very satisfied, as if he were very contented within. He would walk to and fro ceaselessly. Sometimes he sat down, and at other times he was tied in an armchair in a restraining jacket, when agitated. He repeated continually completely incoherent words, all of the most ambitious and extravagant type. A sample of his speech is as follows: "I am the Emperor of Paris; I have been emperor of everywhere; I have lived six thousand years; I am one hundred feet high; I am going to heaven. I am the boss of the capital; I have brought France back to life; I have one hundred thousand golden castles, forty thousand blue gold horses; they go eighty leagues a day; they eat pastry and coffee. I raised myself. I shall never die; I am made of gold; I have twenty-five gold heads; I have eyes like a snake; they are beautiful diamonds." All moral tendencies are obliterated for him. He was incapable of feeling hatred, friendship and so forth. However, at moments, he said his wife's name, and he mistook for her all the women he saw.

Mr. M. stayed several months in this state. Next his agitation grew less; the circle of his ideas grew smaller; he spoke only when questioned and most often replied by repeating the last word of the question asked him.

During the year 1819, he underwent several attacks of cerebral congestion, characterized by sudden loss or diminution of consciousness, loss of speech or difficulty in articulating words, immobility of the muscular system, or convulsions of the face and of the eyes, and trembling of the extremities. Feeling and motion came back to him fairly soon after each attack, but the partial paralysis increased, and he grew mentally more like an imbecile all the time.

In May 1820, he staggered so in his gait that he could hardly stand up; he gritted his teeth frequently; his excretions were involuntary; speech was extremely slow and faltering, and he had three attacks of cerebral congestion accompanied by tremors.

During the month of June he was completely like an idiot; his face was wan and inexpressive, covered with thick sweat, viscous and sticky; he took in no questions addressed to him, or he attempted to make himself understood by means of a few poorly articulated monosyllables. There was no sign of any voluntary act on his part. He could not get up at all, or even sit up. His extremities hung limp from his trunk, and occasionally they would have trembling involuntary motions. Ordinarily he stayed fixed on a sort of chair-bed with a hole under his hips through which he could void (as he did involuntarily) feces and urine. On the twentieth, the cerebral congestion was increased, and a few convulsive movements occurred; he lost consciousness and regained it quite soon, then passed into a kind of general decline. On the twenty-sixth, the heels were darkening and the sacrum was showing excoriation. On the twenty-seventh, there was no trace of sensibility; congestion had set in on the heels which were now blackened and covered with blisters. Toward evening there appeared involuntary movements of the lower extremities, especially on the right side; the respiration was noisy and stertorous, and then death ensued.

During the entire course of the illness there was no variation in the pulse and in digestion, and the patient's appetite was always voracious.

Opening the Cadaver.—Exterior State: The cadaver was 5 feet, 5 inches long, marked by regularity of its general form, good general muscular development, firm flesh, a large scab on the sacrum and gangrene of the heels.

Skull: There is a collection of about 2 ounces of serous fluid between the arachnoidal cerebral layers of the dura mater, which is more abundant on the left

than on the right side. The cerebral arachnoid is slightly altered over the lobes of the brain; over the hemispheres it has nearly completely lost its transparency and is opaque, and grayish white as seen from the exterior surface. Its internal surface is adherent to the cortex of the brain over the whole convexity of the hemispheres and tears off a lining of cortex when one separates it from the brain. It is at least four times its natural thickness and looks like wet parchment. Its cohesion is so much increased that one can lift the entire brain and support it pendant by a slight ribbon of this thickened membrane stripped off the surface of the brain. It can be subjected to considerable stress before it tears.

More than 8 ounces of serous fluid are found at the base of the skull or coming out of the spinal canal. The lateral ventricles and the third ventricle are filled with and distended by this same fluid, which leaps out in a tiny jet when the ventricles are punctured in the region of the optic nerves. These cavities are dilated and a third larger than in the ordinary condition. The arachnoid that covers them is thickened in a remarkable manner and is easy to separate from the cerebral surface. There are extremely thin granulations in the fourth ventricle which can be seen with the eye but cannot be felt with the finger.

The cerebral substance is generally soft; that of the annular protuberance is much more consistent. The superior extremity of the vertebral cord is remarkably hard and cohesive. The arachnoid in the vertebral region is thickened and more resistant than in the natural state.

Thorax: The lungs are healthy. The pericardium contains 1 ounce of darkened blood. The heart is extremely soft and easy to tear; one can easily push one's finger into it. Its cavities are large, dilated and empty.

Abdomen: The gastric mucosa is rough to touch and is neither reddened or thickened. The other organs are healthy.

This observation bears considerable analogy to the case of Mr. A. and shows, as it did, a *délire* that can be attributed only to progressive alteration of the arachnoid. The disease presented three periods during which the mental derangement took in succession the forms of monomania, dementia and finally idiocy, following in a striking way the steps in the progress of a paralysis that from an incomplete state became general. At the beginning and in the first period of the disease a profuse bloody flux was checked. Cerebral congestion then showed itself by trouble in pronunciation and in walking, frequent states of elation, derangement of mental faculties, and a *délire* centering around a certain number of objects. In the second period, the gait was extremely staggering; there was considerable alteration of speech, and his words were very much confused. The excretions became involuntary; the patient was agitated and subject to periods of excitement; his ideas were completely incoherent; his attention could not be gained, and he lost his judgment, reasoning ability and his memory. During this period the patient was the victim of several recurring attacks of cerebral congestion accompanied by a loss of consciousness, and very often tremors and spasmodic movements of his face and extremities. With each one of these attacks the paralysis increased, and his understanding rapidly failed. Finally, in the last period there was no sign of any voluntary action; the paralysis was nearly complete and so very widespread that it included his entire motor apparatus; he was aphonic or only emitted inarticulate sounds in a sort of automatic way when spoken to. He could no longer walk, stand or even sit up; he was in a continually stupid state, with slight involuntary movements of his extremities and at the same time complete idiocy, with no indication that he might have or keep any idea.

The parallel that can be seen at all stages of this man's disease offers us a constant relationship that can be drawn between the *délire* and the paralysis. One can see that parallel running through each of the degrees that separate slight mental disturbance from total destruction of the mental faculties, during the time that this man went from simple or slight motor difficulties to practically a state of total paralysis of all the organs of his motor apparatus. I do not see how one can refuse to admit that these two orders of phenomena are symptoms of the same disease, that is to say of a chronic arachnitis. This last disorder must bear an equal relationship with the physical and with the mental disturbances that it brings about, or at least, that is how it appears from the observations that we are here analyzing. In fact, in comparing this last with the preceding observation, in spite of a great number of points of resemblance, one is struck by several important differences; one explains them most satisfactorily by the observations resulting from the autopsy of the cadaver. In the case of Mr. M. the incomplete paralysis ended in nearly total destruction of his movements; the mental disturbance that began as dementia ended in idiocy; the agitation was never continuous or very violent. In the case of Mr. A. the paralysis was limited (during the main part of the course of the illness) to rendering pronunciation difficult and the gait staggering. The délire, which was at first monomaniacal became maniacal, and the agitation in the second period was extremely violent and convulsive. These symptoms prove that, in the first patient the brain was more compressed than irritated and that in the second patient it was more irritated than compressed. At the autopsy we saw that in the former the arachnoid was so thick that it looked like parchment, and so coherent that the brain could be raised, when held up by one of its shreds, without this shred tearing. We also saw such an enormous quantity of serous fluid in the lateral ventricles that it spurted out in a jet when the ventricular cavity was punctured. In the other, on the contrary, the arachnoid, though greatly thickened, could not support the brain's weight or withstand any strong pulling. In this case the serous fluid was much less abundant. In both instances, however, the arachnoid was adherent to the gray substance of the brain. It was to this cause that we had attributed the spasmodic agitation of Mr. A., but we just noted that this last condition was not present in the case of Mr. M., whose arachnoid also was adherent to the brain. This contradiction is only apparent. In fact, one recalls that this patient was taken several times with set-backs in the form of cerebral congestion, accompanied by tremors and convulsions. And in what way may these symptoms be said to differ from the spasmodic agitation of the other patient? Their nature seems similar, but, in the cases in which they occurred, in one the trouble was continuous and in the other it was spasmodic. The first instance indicated an active irritation and inflammation of the brain, which disappeared after a very short duration. The second was the sign of an analogous irritation, which was constantly maintained in the same organ.

FOURTH OBSERVATION: CHRONIC ARACHNITIS. (MONOMANIA, MANIA)

M. Pierre-Francois-Louis de M., 40 years old, a well built fellow, of a naturally easy-going temperament and a rather self-satisfied manner, somewhat vain, but good on the whole (born of a mother who is now insane), has been for a long time indulging in masturbation. He had a few apoplectic attacks, following which his intellectual processes, especially his memory, have gradually become weak. The last "stroke," which dates about a year back, produced a most marked change in his mood. However, the patient continued working in a legation and kept up

his ordinary work, except, however, for the last fortnight, during which time he has been completely delirious.

He was brought to Charenton on June 27, 1819, in the following condition: face full and red; well nourished; appearance in general very satisfied and contented; marked impairment of any ability to understand what was said to him. His perceptions seemed vague and of a sort that prevented him from occupying himself with any object in a sustained manner. It was hard to hold his attention while talking to him. He was continually distractible, and his memory was so markedly altered that he could barely recall the names of relatives and close friends. He could handle simple objects in a simple way according to his bodily needs, and the same with familiar objects, but he could not associate several ideas, had no judgment and was incoherent in his thought. His thinking seemed dominated with the typical ideas of an "ambitious *délire*"—he is Francis I, and sometimes Louis XIV; there is no more potent king on earth than he is; he owns immense wealth, numberless diamonds of the greatest beauty, and he becomes angry with those who call him by his real name. At the same time symptoms of incomplete paralysis are present. He has difficulty with his speech, which makes him talk slowly; his pronunciation is difficult and sometimes faltering; he has a very insecure gait, with slight agitation; he talks a lot, most often to himself. He walks to and fro ceaselessly and wanders about in the yards and the corridors, exclusively concerned with his ideas, without being held by any fixed judgment of thought. He appears to have no reasonable motives. He sleeps little at night; has an insatiable appetite and calls for food continually, which he devours in the greatest quantity.

During the first three months the incomplete paralysis increased and the dementia advanced. He was unable to recall the words that serve to express the most common things. He did not recognize his sister and his mother; there was complete incoherence in what he said, with ambitious and grandiose ideas always predominating; however, these were extremely various, according to where he happened to be at the moment. A caterpillar that he found became his "best friend," "his beautiful princess from Germany all covered with diamonds." After this period he became violently agitated and yelled and sang. Sometimes he shouted; sometimes he moved his face continually in a disorderly way, and sometimes he flung his arms and legs about. He had moments of blind fury during which he could hardly be restrained by a strait-jacket or the chair he was tied to. This condition kept up for several months with slight irregular remissions and relapses, and then slowly subsided.

About the beginning of 1820, the paralysis grew more advanced, the dementia became more complete; and he could hardly stand up and dragged his legs when he tried to take steps. His voice changed; words came out very slowly and were badly articulated. The sphere of his ideas diminished and became limited to a few reminiscences; for the most part, his grandiose ideas persisted. Soon after this he lost control of his urinary and anal sphincters, and was unable to walk or even to stand up. His voice had a tremor; his face was expressionless, his eyes were fixed and stupid, and the skin began to break down in a serious way in various parts of his body. He grew rapidly thinner and had a voracious appetite. Toward the end of February he grew very limp all over, had but little appetite, looked cachectic, and was in an adynamic state. On April 1 he had respiratory distress and a feeble pulse, and died.

Opening of the Cadaver.—Exterior Condition: The corpse was generally pale, emaciated like a skeleton, with sores on the feet and the great trochanter and scabs over the sacrum.

Skull: A small amount of serous fluid is found between the dura and the arachnoid; the outer surface of the former is greatly injected. One sees between the arachnoidal layer of the dura and the arachnoidal covering of the cerebrum a false membrane spread out as follows: Over the most part of the left cerebral hemisphere it is at least twice as thick as the dura, and this thickness is a little greater about the middle of the hemisphere and less in the anterior and posterior parts as well as at the base of the brain, where the false membrane is very thin, soft and impossible to separate. Its outer surface is sleek, glossy and white like the dura mater, and is adherent to it by many filaments that are easy to break. It appears to be of a cellular nature. Its inner surface is free, in close contact with the arachnoid, and is wet and soft with serous infiltration and rather reddish. This production is very thin on the arachnoid covering of the right hemisphere. It is hard and very resistant to injuries where it is of increased thickness; it is easy to tear off wherever it is thin. The serous lining at the base of the brain shows little reddish clots here and there. The cerebral arachnoid has become very opaque and has a gray-white color; it is so very thick and resistant that one separates it with the greatest ease from the surface of the brain, to which it is adherent in numerous places. It carries away with it, when it is torn off, small pieces of the cortical substance, which on being uncovered appears soft and reddish. These alterations, which are very manifest on the external surface of the hemispheres, are hardly noticeable at the base of the brain and are not present at all over the cerebellum. The lateral and medial ventricles are full of serous fluid, and the arachnoid which covers them is much thickened, easy to separate and covered with a multitude of granulations that can be felt as well as seen. The pia mater is very red and injected in its smallest vessels. The pulp of the brain is soft and injected.

Thorax: Both lungs are engorged with blood; the right shows at its apex considerable induration, and in the middle of the lung is a cavity that could hold a walnut, which is quite empty. About 2 ounces of serous fluid are found in the pericardium; the outer surface of the heart is infiltrated with the same liquid.

Abdomen: The liver is engorged with blood which runs out freely at each incision. The stomach is considerably contracted in its middle part and dilated at its extremities. The mucous membrane, covered with thick and abundant patches of mucus, is intensely red and shows multiple small black spots here and there. The pylorus is narrowed and small, and under the mucosa one sees a cartilaginous substance which surrounds it. The membranes of the small intestine are reddened, excessively so at certain points; this is more marked on the connivant valvules than between them; thick dark red—almost black—blood is mixed here and there with mucus in the lumen of the intestines.

The symptoms listed in these observations have overlapped each other and are due to inflammation of the arachnoid and the state of the brain; they may be considered in the three periods in which one must present them.

First Period: This patient had several apoplectic strokes, which consisted of a sudden injection of the vessels of the brain and its membranes. These attacks, the effects of which never completely disappeared, were followed by a blood congestion, slight but continuous, which produced weakening of the intellectual faculties and, above all, loss of memory.

Second Period: The cerebral congestion increased; the injected arachnoid grew thick, owing to the presence of blood in its capillaries and in those of the pia mater. It lost a part of its transparency. It compressed and irritated the brain; from that came the partial *délire*, the degradation of the operations

of the mind, the general incomplete paralysis, marked by impediment of the tongue and an insecure gait, and from that also arose the slight agitation. The symptoms followed the gradual increase of inflammatory processes.

Third Period: The inflammation of the cerebral arachnoid acquired a certain degree of acuteness; the blood then, called by an even stronger irritation, ran into that region more abundantly; then the internal surface of the arachnoid became adherent to the surface of the brain, and its external surface emitted an albuminous-serous fluid that became organized into a membrane. The brain, more actively irritated, caused violent agitation with spasmodic motions of the whole muscular apparatus; but, as soon as the false membrane was formed, the inflammatory process diminished, the irritation grew less and the compression of the brain grew much more marked than it had been before; thus the paralysis grew more complete and the mental faculties grew more obliterated. The state of violent agitation that the sick man presented during his last days prevented our attentive observation of the symptoms caused by the gastro-intestinal inflammation shown at autopsy. But these, it seems, probably did not have much to do with causing the patient's mental symptoms, because it is probable that they came on much later in the disease and that they were contributory causes of death, as the patient's decline would indicate, which lasted several months.

FIFTH OBSERVATION: CHRONIC ARACHNITIS. (MANIA, DEMENTIA, IDIOCY)

Louis Levéillé, a soldier of the royal guard, 47 years old, of full-blooded temperament and a robust constitution, was greatly chagrined after having been forced to retire for an act of insubordination which caused a military decoration (*Légion d'honneur*) to be revoked after he had worn it for some time.

He showed signs of mental disturbance, though no one thought much of them until the end of the month of August 1819, when he had to come into the hospital of Gros-Caillou where he stayed for two weeks in a state of *délire*. At times he was calm and tranquil; at times he was greatly upset and broke everything he could get his hands on.

On Sept. 11, 1819, the day he was admitted to the Charenton Hospital, his face was flushed, he looked silly and had an astonishingly contented air. He replied slowly, often by monosyllables, to questions asked him, and showed usually a silly expression and a foolish grin. He repeatedly said that he felt well and was getting on fine, but he had forgotten the most common things, such as the number of his regiment, the name of his colonel and his own age. He appeared to have ideas of grandeur but did not speak much because of his generally decreased intellectual faculties; his gait was staggering; his words were slow and faltering. He slept well. He ate a great deal, with voracity. These symptoms made marked progress during the first months of his stay at the Charenton Hospital. Then they remained stationary. Until the month of September 1820, the patient was in the following condition: sensations very obtuse (no pain even when strongly pinched); a stupid facial expression, vague, uncertain or in a fixed state. One had to speak to him in a loud voice or shout in his ear to fix his attention even slightly to questions asked him; usually he did not understand at all and replied with poorly articulated sounds. His intellectual faculties were obliterated. He had no memory of what had happened to him, or even of what had just happened under his eyes, and he showed a total loss of recollection for his past life. The sphere of his understanding was reduced to a few grandiose notions

that were incoherent and in no way related to facts; he was rich, he felt just fine, he ate well, etc. General incomplete paralysis was very marked; speech was badly interfered with; his words were chopped-up, tremulous and poorly articulated. He walked slowly and with extreme difficulty, dragging his legs. He was very quiet most of the time. He sat on a bench in the boiler-room, with his hands on his knees and his head slightly bent over, in a state of silence and apathy continually, without making any motion. At moments he would get up, would stay a long time standing without changing his place; sometimes he would wander here and there about the yard with no plan or motive.

In the month of September 1820, he can no longer articulate words. When he wishes to speak he makes unintelligible and confused sounds that babble for a long time in his mouth. He cannot walk or even stand up. His excretions are continuous and involuntary. He has to be put into a jacket and tied to a chair with holes for him to void and defecate through, otherwise he would fall out of it. He keeps on eating with a voracious appetite and never is satisfied. He looks well from a general physical point of view.

At the beginning of January 1821, all symptoms are getting worse. He is symptomatically a total idiot. His face looks like that of an animal. His sensations are so impaired that one can pinch him very strongly and he hardly feels it. His eyes are fixed and have a strange stare. He is unable to comprehend the simplest ideas. However, he does turn his eyes a little in one's direction if one yells at him in a loud voice. He is generally paralyzed. He has complete aphonia, and dorsal decubitus; his arms lie motionless at his side, and he never moves them even if they are pricked. He is famished, hungry all the time, and has enormous obesity. Toward the fifteenth, two deep sores develop on the sacrum. These become very widespread and break down, become darkened about the edges and very deep, and from them fetid pus flows freely. At each dressing pieces of flesh fall out without causing him any pain. His pulse becomes strong but not rapid; his voracious appetite continues, and there develops a condition of monstrous obesity, his extremities being one and a half times their natural size. His face is distended by billows of fat, and his respiration becomes greatly labored. Now he cannot make a single motion, but he distinguishes food when it is brought to him and seizes it in his mouth with a sort of instinctively impulsive movement. Of all the faculties of human understanding, there are left to him only a few confused sensations.

On January 20, the sore has spread enormously; respiration is very difficult, and the mouth stays half open. However, the sick man still can eat the soup which the nurse brings him.

On January 25, coma supervenes; the pulse is weak and slow. On the twenty-seventh, death occurs.

Opening the Cadaver.—The sacrum is exposed by a deep sore. The body is extremely obese, fat being 3 inches thick over the thighs and 2 inches over the legs, and moderately infiltrated with serous fluid.

Skull: The dura mater is distended. On opening this membrane there spurts out about 12 ounces of serous fluid, slightly blood-stained. This fluid had kept spread apart the two layers of the arachnoid and was occupying and overlying the anterior and superior parts of the cerebral hemispheres. When it had all run out, the dura was much too large for the brain, and collapsed with wrinkles and folds over the surface of the brain. The sulci were widened and deepened; the convolutions were shrunken and flattened, and the hemispheres were so extremely compressed in the frontal parts that they looked not round but like a triangle

whose base was posterior and whose apex lay anterior. Comparing the lateral spread or width of the superior surface of the brain at different points with that of the cranial vault that should correspond to it, one sees that on the posterior surface the brain filled the vault exactly, while on the anterior cerebral surfaces there is cerebral substance missing and leaving a gap between the brain and the cranial vault, at least an inch in distance, under the frontal bones on each side.

There is a false membrane over all the arachnoid, except in the occipital region and over the cerebellum. There is a thinning of the arachnoid in certain places; it is increased in thickness in other places and is separated with ease from the arachnoidal tissue of the dura mater with which it is in contact on its external surface. Its internal surface, reddened, shows bright red points here and there. The arachnoid of the dura mater is greatly injected.

The external surface of the cerebral arachnoid shows very small blood clots on the lateral aspects of the hemispheres. This membrane is thickened and a little more resistant than in the natural state over the anterior and superior parts of the cerebral hemispheres, where it is adherent to the cortical substance in some points. It is stuck to itself in the great fissure and injected over all the surface of the brain except the cerebellum. The pia mater is red, injected with a great deal of blood which runs down into the sulci when the pia is torn off. The lateral ventricles, full of serous fluid, show granulations on their surfaces. The cerebral substance is healthy.

The abdominal and thoracic viscera are buried in deposited fat. The stomach, greatly contracted, shows voluminous wrinkles of its mucous membrane, which is slightly rosy.

It is regrettable that we have so little information about the predisposing and occasioning causes of this disease and about the condition of the patient before he came to Charenton. We know only that he had been in a state of mania, with moments when he was in a furor. One sees, by the preceding observation, that Levéillé stayed a year in a state of general incomplete paralysis, characterized by an extremely staggering gait and very difficult pronunciation, accompanied by complete dementia, with incoherent and vague ideas and with no sign of agitation. His last period was marked by nearly complete insensibility in all spheres, the destruction (practically complete) of all ability to move his muscles and as complete idiocy as one can think of. The preceding observations have proved that these symptoms have resulted from compression of the brain, and as the latter symptoms occurred in the case of Levéillé with greater intensity than in the cases of the other patients, reasoning it over makes us think that the compression likewise must have been more considerable. Necropsy has fully confirmed this induction in showing us the brain placed between two very strong compressive forces, i. e., on one side a great quantity of serous fluid compressing the ventricles, and on the other side about 12 ounces of this same fluid, which, weighing down the superior region of the brain, had flattened out its convolutions and deepened and widened its sulci. One must attribute the agitation of the first period to the most acute state of the patient's frenzy, at the period when the arachnoid, which had reached its highest degree of inflammation, produced, along with serous fluid, albuminous flakes that slowly became organized into a false membrane. Later the irritation grew less; the serous exhalation grew progressively worse, and it all ended in producing a symptomatic hydrocephalus in the same manner that pleurisy and chronic peritonitis give rise to the various stages of dropsy that follow these conditions.

SIXTH OBSERVATION. CHRONIC ARACHNITIS. (MELANCHOLIA, DEMENTIA)

Mr. R., a priest, aged 56 years, of a "lymphatic" (heavy, dull) temperament, from a healthy family, was working ceaselessly, with sustained application, at the most abstract studies, when, in the middle of the year 1816, he gave signs of disturbance of his mental faculties. He had frequent lapses and often lost his memory of events that had just happened under his very eyes; he himself became aware of his "weakness in his head" and "trouble with his ideas." Then the chagrin he felt on finding himself in such a state threw him into a profound melancholia that lasted about six weeks. After that his faculties failed rapidly, and it did not take him long to fall into a state of semiparalytic dementia, accompanied by the following symptoms: a heavy and befuddled head and frequent complaints of feeling severe pains therein, very circumscribed ideas, which were vague and incoherent, and complete loss of memory to the point where finally he could not recognize the maidservant who had cared for him for eighteen months; very insecure gait. He was habitually calm.

On May 12, 1818, he was brought to Charenton Hospital; at this time his face was pale and rather swollen; his speech was a little encumbered; there was a tremor in his words and an air of general suffering about him; he made many complaints, and his ideas were very disconnected and entirely irrelevant, with no special trends predominating. His gait was vacillating; at times he would cry, and he lost his urine and feces involuntarily. He had good appetite and frequently wanted to eat.

From this time on to the end of the year these symptoms increased gradually without much change in type. The patient became weaker every day, and on two or three occasions had spells of loss of consciousness; but this always came back, a little after the shock, though the general paralysis made continual progress.

On Jan. 4, 1819, he did not respond to any question made to him; he paid no attention at all to what was around him; his loss of memory was so complete that he could not recall the name of the simplest things, like bread, which he now named *religion*. His own name, that of his parents, people who worked for him, God, etc., were all equally gone from his mind, and if at some moments he did appear to retain some memory of these objects he fell back very quickly into his usual state. The sphere of his ideas was extremely limited; they were often without any coherence and became related principally to his primary needs. Ideas did present themselves occasionally to his mind but were slow and with no coherence. He gave out a few opinions at times, but these were extremely simple and related to his own bodily care, some instincts of which had survived the general degradation of his understanding. Usually he did not reply or replied in monosyllables. His words were very slow and his gait so staggering that he could hardly stand up at all. He fell down frequently, and his excretions were involuntary. Usually he stayed tied to an armchair with a hole in the seat through which he could void and defecate, or he stayed in the yard of his ward where he would try to take a few very unsteady steps, clinging to the wall or to trees to keep from falling, wearing a facial expression that combined obliteration of his faculties and an air of suffering; he complained from time to time. Often he said the words *my friend, my poor friend, sick man,* etc. He was tormented by a continual appetite and often asked for food.

On March 31, his face was very pale and expressionless and swollen; he could not stand up; he complained continuously, was generally extremely weak and grew progressively thinner and thinner. He always wanted to eat, and yet he was in an obvious decline.

During the month of April a continuous diarrhea was present among other symptoms. He grew progressively weaker, and by the first of May (the diarrhea continuing) he was in a state of complete prostration, extreme emaciation, and total dementia. He gave no response to questions or any sign that indicated they had made any impression on him; he had an aphonia, but from time to time he emitted confused complaining cries. On May 22, his weakness was carried to the last degree; his pulse was slow and very weak, the respiration was shallow and labored, and on the twenty-third he died.

Opening the Cadaver.—External Appearance: The cadaver was in an extremely marasmic state.

Skull: There is collected a great quantity of serous fluid at the base of the brain and in the vertebral canal. About an ounce of the same fluid is found between the two layers of the arachnoid, and this membrane is whitish and opaque at several points, though quite transparent in others, where through it can be seen considerable serous fluid which gives it a gelatinous appearance. It is thick and very resistant and can easily be taken off the surface of the brain. Its outer surface shows a large number of excessively thin granulations that the eye can discern only when it is held in certain positions. The pia mater, very red, is infiltrated with a great amount of serous fluid that runs out from all parts and fills up the sulci. All these changes in the meninges are noted principally over the convexity of the hemispheres of the brain. One can see under the base of the brain mass, under each median lobe of the brain, an irregular pouch, transparent, soft, having an elongated form, larger and more distended in anterior than in posterior dimensions, free on its under side which is lodged in the lateral and medial fossae of the base of the skull, and adherent to the brain on its upper surface. It was filled with a limpid serous fluid, and one could see that inside it were filaments of cellular tissue which were interlaced in various directions, the areolae of which, infiltrated with the same liquid, had a gelatinous appearance. Above this pouch a slightly thickened membrane separated its cavity from that of the lateral ventricles, and the cerebral substance that ordinarily forms the floor of the ventricles had entirely disappeared. This partition was pierced in several places, in such a manner that there existed a communication between the cavities of what was left of the ventricles and the cavities of the serum-filled pocket. The cerebral substance against which the pocket had been pressed was softened and had been broken down into small whitish areas. The lateral ventricles, vastly enlarged, were enormously distended by the serous fluid. The rest of the brain was healthy.

The chest and abdomen were not opened.

Considerations.—This observation does not differ from the one before except in its inception and the degree of intensity of its symptoms, which were less extensive than those of Levéillé. He did not have at the start, like the preceding patient, nor at any epoch in his sickness, any agitation or furor. The dementia developed early and lasted throughout the course of the disease. Complete idiocy was present only during the last few months of the disease. The general incomplete paralysis increased up to death, but in this case the patient did not come to the extreme stage of callousness and immobility that made Levéillé the most striking picture possible of a living automaton. The compression of the brain, judging by the alterations found at autopsy, must have been excessive. One can hardly explain the formation of the pouch or pocket that existed at the base of the brain unless one might claim that an accumulation of serous fluid occurred first in the lateral ventricles and that after it had extended beyond the limits of

the walls of these cavities it had worn out, little by little, the cerebral substance that formed the inferior floor of the lateral ventricles, and finally destroyed it completely, and piercing the ventricular arachnoid it had infiltrated into the web of the pia mater of the base of the brain, had detached the arachnoid that covered this part and given it the form of a pouch.

Such decay could not have occurred without the brain having had to meet conditions of extreme compression, and with this condition present one can only wonder why the obliteration of the patient's intelligence had not occurred sooner, unless it is due to the fact that the principal alterations may have occurred in the last months of the patient's life. As to the absence of agitation, one can conceive that the inflammation of the veins of the arachnoid gave rise to the thickening of that membrane, having occurred in a slow, not marked manner, and having caused, from the very start of the disease, an abundant emission of serous fluid; the brain may not have been irritated.

RÉSUMÉ OF THE FOREGOING OBSERVATIONS

My plan, in publishing the observations just read, was to throw some light on the nature of mental diseases by making chronic arachnitis known as the cause of a kind of symptomatic psychosis that until today has been confused with "essential" psychotic states. The intention of tracing in a general way the history of that disease also has been as far from my intention as it is beyond my ability. Let us hope that Professor Royer-Collard, who has been observing this disturbance for a long time in Charenton Hospital and who has come to possess most valuable material concerning it, will finally share with the public the fruit of his experience and his observations concerning this disorder which, by its frequent occurrence, its symptoms, its progressive nature, its gravity and its final results, nearly always tragic, and its wide social complications, deserves to have fixed on it the attention of the entire medical profession. I shall limit myself to the following propositions, which are corollaries of the preceding observations, and of the reflections that accompany them.*

1. The arachnitis, or chronic frenzy, is a disease that differs in an essential manner from acute arachnitis, of which it is not necessarily an end-state or termination.

2. Concerning the cause of this, one finds many predisposing and occasioning conditions, but each one of them acts only by drawing the blood to the head and causing it to accumulate in the vessels of the pia mater and of the brain. The cerebral congestion, slow or sudden, that results from this is the next and necessary step in causing the illness. It works on the tissues of the arachnoid.

3. This chronic frenzy consists in alterations in the arachnoid which are quite analogous to those of acute arachnitis; except that in the case of chronic arachnitis there is no suppuration, while this commonly occurs in acute arachnitis. The anatomic characteristics of this chronic inflammation are: opacity of the arachnoid, thickening of it, which may be quite slight, or equal to or thicker than a sheet of parchment, an increase in its cohesive and resistive qualities between limits of extreme variation, which is sometimes so considerable that the membrane cannot be torn and can support the weight of the whole brain without tearing, and the production of a large amount of fluid that collects at the base of the brain, infiltrates the tissue of the pia mater and crowds into the lateral ventricles which it

* They are equally based on a great number of other observations on the same disease that I have collected.

may distend beyond the natural limits of their walls, thus producing a symptomatic hydrocephalus; quite often there is adherence of the arachnoid to itself and to the exterior surface of the cerebral hemispheres, in more or less extensive regions, and quite frequently there is injection of the pia mater and thickening of the arachnoid of the ventricles, over which one can see delicate granulations visible to the eye and sometimes considerable enough to be felt with the finger, in a certain number of cases; sometimes there are false membranes and, rarely, collections of hemorrhagic fluid between the two layers of the arachnoid.

4. The symptoms of chronic arachnitis can all be included in the conditions of general and incomplete paralysis and derangement of the intellectual faculties. These two types of alteration usually advance with equal step in the progress of the disease and enable us to divide the disease into three stages. In the first, pronunciation is noticeably impeded, the gait is unsteady, the disorder of the patient's understanding is manifested by a weakening of his intelligence and a monomaniacal *délire* that more or less dominates the sick man, who is often in a more or less considerably elated state.

In the second period the disturbance of speech and the partial paralyses of the arms and legs may remain in the same condition they were in the first stage, or they may become much worse. The *délire* remains maniacal and general, frequently accompanied by dominant ideas; the patient may be agitated, which condition varies according to his volubility and mobility; this causes him to be restless, move from place to place until sometimes his furor is completely violent and uncontrollable. Finally comes the third period. It is, in general, characterized by a state of dementia and an increase in the general paralysis, which may be partial or complete. The words of the patient are faltering, tremulous, very difficult and sometimes unintelligible. The gait is vacillating, very staggering, or even impossible. The excretions are involuntary. The understanding is extremely weakened; he retains only a few vague ideas that are usually incoherent and more or less fixed, and it is in this stage that he becomes calm or immobile, though at times he may have periods of more or less severe agitation. This stage usually ends in a state of complete paralysis of all voluntary movements and of complete idiocy. Each patient does not regularly present these three periods. It is not unusual to see one omit the second; the latter sometimes offers spasmodic agitation that may be continuous or periodic, and quite often, in the third stage, attacks of cerebral congestion occur, accompanied by loss of consciousness, sometimes by tremors or convulsive movements, and followed by an increase in the symptoms of the disease, even epileptiform seizures.

These are the most essential features of chronic arachnitis or chronic frenzy. I have not room here to speak in detail of the whole picture, or to discuss its progress minutely, or the ways it terminates, or all its signs, or the methods of treatment that can be applied to it. I shall have attained the objective I set out for if this part of my work can prove that *chronic arachnitis does exist and that it is the cause of a symptomatic mental disturbance.*

ESMARCH AND JESSEN'S THEORY THAT SYPHILIS IS AN ETIOLOGIC FACTOR IN MENTAL DISORDER

However much Bayle may have accomplished in making clear the clinical symptomatology and the pathologic processes in chronic arachnitis, he, perhaps wisely, did not commit himself as to etiology. While it is true that in some of his cases, for instance in the first and second

cases, he spoke of venery and syphilitic disease, he offered this only in conjunction with other possible etiologic factors and laid no special emphasis on any particular cause. The first important treatise dealing with syphilis as a cause of mental disturbance is to be accredited to Esmarch and Jessen. Their contribution on syphilis and mental disturbance was published in the *Allgemeine Zeitschrift für Psychiatrie und psychisch-gerichtliche Medizin.*[6]

Syphilis und Geistesstörung.

Von

Dr. Fr. Esmarch und **Dr. W. Jessen.**

Die Bemerkung, dass Syphilis Geistesstörung (nicht etwa bloss hypochondria syphilitica) hervorbringe, findet man von guten Beobachtern, von Vidal, Simon, Knörre u. A. häufig ausgesprochen. Um so auffallender ist es, in der speciell psychiatrischen Literatur so wenig über die Syphilis und ihr Verhältniss zu den Psychosen erwähnt zu finden und wir müssen nachdem wir, einmal aufmerksam geworden, in kurzer Zeit drei zweifellose Fälle dieser Complication beobachtet, vermuthen, dass dieselbe bisher oft übersehen wurde. Um hierauf die Aufmerksamkeit zu lenken, theilen wir jene Fälle im Nachstehenden mit.

***A.*, 41 *Jahre alt*, *Advocat*. Erbliche Anlagen zu Geisteskrankheiten fehlten; psychisch war das Gedächtniss besonders entwickelt, der Verstand von mittlerer Stärke, die Gedanken aber mehr aneinandergereiht, als logisch verbunden; grosse Gutmüthigkeit, schwache Willenskraft bildeten die Hauptgrundlagen des Charakters. Die körperliche Constitution war vortrefflich, er war frühen und zahlreichen Excessen in Venere zum Trotz ausser erfolgter Ansteckung niemals krank gewesen und hatte, seit 14 Jah-**

Fig. 3.—Facsimile of the first page of Esmarch and Jessen's work on syphilis.

Their conclusions are based on the analysis of three cases of mental disturbance in which they believed that they had found conclusive evidence of syphilis in the patient, both in symptomatic expressions of the skin or mucous membranes and in the effect on these lesions of antisyphilitic medication. From a reading of the report of their cases it is difficult to decide the exact classification that these cases should occupy

6. Esmarch and Jessen: Allg. Ztschr. f. Psychiat. **14**:20, 1857.

in the neurosyphilitic hierarchy; that is, whether they should be considered as meningovascular cases, or as cases of dementia paralytica. However, the importance of the contribution lies in the fact that Esmarch and Jessen called the attention of the medical profession to the theory that syphilis can produce mental symptoms and that these symptoms may not respond favorably to antisyphilitic medicaments.

No better criticism can be offered than their own modest summing up of their contribution. "Three cases, naturally, could not determine the difficult question whether this is cause or complication, and it must be admitted that syphilis can also occur as a chance complication of the psychic disturbance, since mentally diseased patients can become infected and later have constitutional syphilis. We wished, however, only to express our belief concerning the aforementioned cases and to stimulate similar observations and also differences of opinion." Unfortunately, however, their desire to stimulate observations and to arouse interest in the subject was not very successful. It was not until many years later that the work of Fournier, in France, Kraft-Ebing, in Austria, and Erb, in Germany, produced the desired result. Nevertheless, to these authors must go the credit of having been among the first, if not the first, to have given material to support the hypothesis that syphilis is a frequent cause of mental disorder.

TRANSLATION OF ESMARCH AND JESSEN'S ARTICLE "SYPHILIS AND MENTAL DISTURBANCE"

The observation that syphilis produces mental disturbance (other than hypochondria syphilitica) has been frequently stated by good observers such as von Vidal, Simon, Knorre and others. It is striking that so little is to be found in the special psychiatric literature about syphilis and its relation to psychosis. And since we have recently observed three unquestionable cases of this complication which appeared at one time, we must suspect that this observation has often been missed. Therefore it is to relate these observations to this idea that we report the following cases:

A., a lawyer, aged 41, had no hereditary disposition to mental disease. His memory was especially well developed. He was of average intelligence. His thoughts were more sequential than logically connected. He had excellent spirits, and a weak will. These factors form the principal character-symptoms. The bodily constitution was excellent. Formerly he had indulged excessively in intercourse, and in spite of resulting contagion he was never sick. He had married fourteen years ago and had five healthy children. From the time of his student years till his marriage he had had numerous attacks of gonorrhea and chancres, but these he had kept so concealed that we obtained our first information of them when the secondary symptoms directed us to a more exact investigation. A case of gonorrhea contracted before marriage had been taken by the house doctor for an unsuspected case of catarrh.

For a considerable time the patient had been out of sorts and in contrast to his natural good spirits. For a number of months he had been definitely melancholy, monosyllabic and immobile. He had been oppressed by unnecessary worries

over his nutrition. At the time of his admission to Hornheim, in January 1854, there were to be noted neither any abnormal ideas nor any bodily ills, but a very definite abulia. He slowly became active and very voluble and soon went past his normal vivacity. He prattled a great deal about his travels and kept snapping his jaw in a very unusual fashion; in May he was so excited that it was necessary to limit his freedom. (He was given sodium nitrate, digitalis and rain baths.) Soon there occurred sleeplessness, restlessness, flight of ideas, disorderly behavior and a few outbursts of violence, which made it necessary to isolate him in a cell by the end of May. While he was there there developed on the anterior surface of the left shin a phlegmonous inflammation which, the patient explained, had been the result of a fall over the root of a tree in the neighboring woods. This became full of pus and left behind a round ulcer that assumed a definitely syphilitic character. It showed mainly induration and sharp demarcation of the border, a darkly stained border part and a red-brown coppery center. The anterior surface of the tibia itself, in the region of the swelling, appeared to contain irregular nodules. Very quickly there appeared in this region two pustules which were covered with thick brown scabs, under which there rapidly appeared a round ulcer. Even though there were no symptoms of secondary syphilis to be found in the remainder of the body, the nature of these lesions left no doubt. At the time of examination on entrance, the patient himself admitted that there had been syphilitic lesions on his genitalia, and his references, according to confiscated papers, confirmed the diagnosis readily enough. Through uninterrupted observation it was possible to keep the patient in bed and on a strict diet (only soup or bouillon and vegetables). Bathing with warm water and sublimate in solution by mouth were prescribed. Of the last he received from July 14 to September 15, 13 grains; then, as salivation occurred, he received potassium iodide, until October 31, to the amount of 15 ounces. During this treatment the ulcers gradually closed and the syphilitic symptoms improved. Toward the middle of August there occurred in the region of the liver, on the right side, a very definite but uncharacteristically colored and terrifically painful herpetic lesion, with many large confluent pustules which left behind rather deep small healing ulcers. On September 21 we allowed the patient to be up as bodily recovered. The psychic ills had likewise vanished except for a lasting absence of recognition for the past period of illness. The patient was dismissed on Jan. 14, 1855, after his relatives had found him absolutely normal.

During the winter and spring and up until the summer his condition was considered satisfactory, since the patient had devoted himself to farming and lived among people who were in no position to recognize the state of his spirits. So, during this time, there was no observation as to whether a depression occurred, which in any case could not have been significant, at least, until the time that his excitement had recommenced. Toward the end of the summer he began to prattle, to snap his jaws and to carry on in a bizarre fashion. For example, he would slide around on his buttocks in his room and would sit on a hummock alongside of the highway and shout out the names of the neighboring villagers. He ordered for himself twenty newspapers. The next occasion for his transfer back to Hornheim was a disturbance which he had caused by carrying out an Italian monolog in the theater.

His condition was exactly similar to the excited state of one year before, except that at this time there was a serious catarrh, with coughing and sneezing, which he had carried over from a serious cold he had had in September. On one rather reddened area there appeared small blebs, but otherwise there was not a single trace of syphilitic infection. In the interim he had had no desire for coitus. On

the hypothesis that the troubles mentioned might be a syphilitic infection of the brain and mucous membrane, we gave him (besides a supportive diet) sublimate, and from January 4 to 24, 18 grains of this drug was given with egg albumin. On the tenth there occurred vomiting and diarrhea, so that the treatment had to be withheld for a day. On the twenty-fourth there appeared the first traces of salivation. In the meantime, however, the mucous condition and the excitement had completely disappeared, and the specific treatment was broken off. He maintained, however, a marked disposition to catarrh, especially of the lungs and also of the stomach, with vomiting. Though he recognized his bodily complaints completely, he unwillingly admitted his mental symptoms, though he finally did, more as the result of our persuasion than through his own insight. He returned to Hornheim in August 1856, and has shown for the last few weeks a depression of spirits to which external causes apparently have not contributed.

In this case it appears that the two coincident occurrences of symptoms of bodily and mental disease and the quick and definite action of mercury indicate that syphilis was the cause of the psychic disturbance. The remission and the still incomplete eradication of the disease process do not, according to our opinion, contradict this, since it is well known that constitutional syphilis cannot be eradicated with certainty by the use of the mercurials.

B. was 36 years of age, a manufacturer. No syphilitic taint was noted. The patient distinguished himself through his great sophistication in regard to materialistic affairs. He possessed a very keen intelligence, which was augmented by his manifold experiences. He was very energetic, yet in fact was a sympathetic fellow. He did not have a very well rounded personality. He had a very great self-esteem and a desire to be dominant through his intelligence and sophistication. His total character, therefore, was never completely developed. In the beginning a lawyer and already a court referee, he was forced in his twenty-sixth year (1844), on account of pressing circumstances, to take over a factory which had fallen into difficulties, and in a short time he made himself known in England for his factory products. He prospered in his business and led it successfully through the dangerous revolutionary year, leased it in 1850 and has lived on the profits since.

As early as 1846 he had written a pamphlet in verse of mystic content, with quotations from the Apocalypse, and already he spoke frequently of his enormous capabilities, which were to let him easily and quickly discover the principle of perpetual motion, the quadration of the circle and so forth. Many other pamphlets followed, all of which purported to bring about a complete social transformation of the world through the sentence "Geld gleich Werth." These pamphlets contained here and there, in spite of their enormous deficiency in logic, so much cleverness that prominent French national economists referred to them and were led to disprove them! Frequently, he had them published in three languages and had them sent in large numbers to prominent people. The time that these fantastic ideas became pathologic—or whether they were so from the beginning—cannot be stated, but they appeared to become ever more firmly implanted in his thoughts, and his gloominess of spirit became steadily greater.

He did not remember when he had first contracted syphilis. He had been treated incompletely by some university friends, and not until later had he taken himself to a reputable doctor. Toward the end of the year 1849, he had noted that after an upset he had found on the following day that his tongue was "heavy" and his mouth drawn to one side. The physician had him bled, and he was up and around after a few days, but at the same time there appeared secondary syphilitic exanthematous ulcerations of the mouth and swellings of the testes.

After a long use of the Zittmann and Dzondi treatment these symptoms disappeared, but the chronic inflammation of Schneider's tunic and of the periosteum of the nasal bone and muscles persisted for a long time. Then there developed a carious lesion of the alveolar processes of the maxillae, as the result of which both anterior incisors were lost. By the use of yellow mercurous iodide and sarsaparilla, the progress of the disease was checked, and in 1853, the patient went to Paris, to Dr. Ricord, who removed a piece of bone from the carious place in the maxilla and gave him potassium iodide. In the autumn of 1853, he returned, apparently greatly benefited.

The psychic disease, however, had not been eradicated. He had written much in Paris, and now expected that surely his pamphlets would stir up immeasurable interest and would reform the world. He sent them to a very prominent statesman and proposed at the same time to the statesman's daughter, whom he had seen only once. Then he furnished a room in his apartment in a very grandiose manner. In the evenings he would light all the candelabra of this room, etc. In February 1854, he again returned to Paris, where his friends found him greatly elated, and in March he went to London, where his mental disturbance broke out in an unmistakable manner. He mistook those around him for Palmerston, Macaulay and others. He mistook his housemaid for Lady Palmerston, and believed that these people were spying on him and that they were attempting to ruin his "mission." He had visiting cards printed and had put on them the name of the aforementioned girl as his wife, and also had made a seven-sided seal engraved with mystic letters.

He was admitted to Hornheim on April 25, 1854. At first he appeared in good general physical condition. The syphilis appeared to be extinguished, but the psychic abnormalities were marked. In spite of his sharpness in perceiving detailed matters, there was a definite incoherence of ideas. His self-complacency was excessive, and his delusions of persecution with numerous illusions became more and more definite as time went on. He thought that enemies kept him in prison. The physicians were in the enemies' service, and had to carry out their commands even against the physicians' will. His mission was to be interfered with, and he was to be destroyed bodily and spiritually. His marriage was to be prevented; the conception of a child, a social liberator and a messiah, was to be stopped. His enemies made him impotent, and so forth. His own deliberate interpretation of experiences or words, which he called "symbolization," gave him these ideas. For example, when there was discussion of mathematical roots, it "symbolized" to him that the "root" should be taken out of him, that is, that his potentia or ability to conceive would be removed.

On July 8, he attempted to escape. As a result his window had to be barred and thereupon he began to complain that the bars caused him double vision. From the very beginning his eyes had never moved symmetrically on the optic axes, and this appeared to be a congenital failure; but from now on a divergent strabismus of the left eye became more and more severe. Any examination became almost impossible since the patient, instead of paying attention, wished to do the examining himself and during the subjective parts of the examination, as he himself later admitted, intentionally gave wrong answers "to put off the enemies." Therefore from the beginning it was very difficult to bring to light the exact nature of his trouble. But the squint became more marked, and the pupils began to widen and became immovably fixed in the outer angle of the eye. Therefore it was not entirely evident whether the function of the left abducens predominated over the paralyzed oculomotorius or not, or if, which was highly possible, there was a

progressive pathologic process, syphilitic in origin, in the interior of the brain, causing the symptoms. At the beginning of the symptoms of paralysis we had stopped the treatment with tartrates and rain baths, which the patient had been receiving because of heat and pressure in the head, and we later ordered potassium iodide to which therapy we were limited exclusively in the future, since the patient was not without some knowledge of chemistry and would allow no unknown medication to be used, least of all mercury. For the long period from August 7 to October 19, and with apparent success, he received a total of 24 ounces of potassium iodide and 10 ounces of ammonium iodides. In spite of this the disease progressed; the eye became immovable; the pupil, wide; a ptosis occurred, and toward the end of October there appeared sleeplessness and precordial discomfort. As a result of these there appeared in the psychic realm, in place of the previous elation and overactive behavior, an increasing dulness and bad disposition, with worrying over bodily symptoms which the patient recognized clearly and made even more painful through his terrible descriptions of them. He complained that he was to be blinded intentionally, that he was being martyred, that his brain was being eaten up by secret poisons and that, in fact, they intended to lash him, castrate him and so forth. To provide him with some sedative and quiet, a palliative drug, namely morphine, was used.

The disease made rapid progress after the middle of January 1855. The lens became opaque, and in the middle of February the ptosis was complete and deafness occurred in the left ear. From the beginning of February there were vertigo, gastric distress, anorexia, coating of the tongue and constipation, for which laxative drugs and also emetics were given at the earnest request of the patient. Very quickly the paralysis extended to the right eye and caused the cornea to be fixed outwardly, the pupil to be widened and a half ptosis to occur. Finally spontaneous vomiting and diarrhea occurred, with death on March 7, 1855.

The autopsy, which was performed in our presence forty hours after death by Professor Weber, gave the following interesting results: The body was well developed, the thorax broad and domed; there was a strong musculature with large deposits of fat; rigor mortis was present.

Head.—The scalp is thick; small ecchymoses are visible in various places in the subcutaneous tissue. The calvarium is moderately thick, the diploe poorly developed and rich in blood, and on the inner surface there are no exostoses. The dura mater is rich in blood and on the anterior surface of the pars petrosa there is a thickened space, the size of a bean, infiltrated with exudate and of a red-yellow color. The arachnoid on both hemispheres is opaque and thickened, with many white spots of exudate, the size of the head of a pin. Pacchionian granulations are noted; they increase in number toward the central longitudinal sinus. Under the arachnoid a definite serous fluid is present (chronic arachnitis). On the base of the brain, in the region of the pons and the medulla oblongata, the arachnoid is firmly adherent to the dura mater. The point of the infundibulum, along with the dorsal side of the chiasm, is also firmly adherent. The right oculomotor nerve, which leaves the brain with a normal thickness, is swollen to 4 mm., which is three times its normal thickness. It is reddened and turned into a lardy mass of rather firm consistency. The left oculomotor nerve is likewise deteriorated along the whole length of its course in the cranial cavity and is irregularly swollen even to six times its normal thickness.

The left trigeminus nerve is likewise thickened; the right trigeminus is normal, and the right abducens is significantly thickened and slightly reddened.

On the left side, between the facialis and the acusticus, there is a large extravasation of blood, and the acusticus itself appears definitely thickened and of a dirty yellow color. All the other cranial nerves are normal. The basilar arteries and the arteries cerebri profundi show on their inner surface small atheromatous deposits. In the left arteria cerebri profunda, close to its branching from the basilar, there is a round yellowish plug of fibrin which fills out the lumen of the vessel quite completely and is firmly adherent to the wall. Behind the plug the vessel is obliterated to a mere cord and, corresponding to this, the left thalamus contains an old, brown red area of softening in the middle of which there is a cyst the size of a pea filled with a serous fluid.

The eyes could not be examined further.

Chest and Neck.—On both sides the tonsils are somewhat hypertrophic and indurated with deposits of connective tissue.

In the apex of the left lung a small tubercle is found; otherwise the lungs and the heart are normal.

Abdomen.—On the surface of the liver there are white stripes which, when cut, appear to be a partial thickening of the peritoneal lining and partly a superficial contracting scarring of the otherwise normal parenchyma. The spleen has grown to the lining of the peritoneum through old adhesions and is rather poor in blood, but of normal consistency. The kidneys are of normal size, with firm parenchyma; the tunica propria shows partial thickening, and when torn off, takes pieces of the cortex with it in certain places. Otherwise the abdominal organs are normal.

In the tunica vaginalis of the right testis, there are about 6 ounces of serous fluid. Both testes are rather thickened and indurated. On incision there appear in numerous places small rather hard masses of exudate of a yellow or a yellow-gray color, which is apparently consolidated pus.

Subsequent detailed examination of the degenerated oculomotor nerve gave the following: On cross-section, the whole nerve substance, including the nerve fibers, appears to have been changed into an intensely reddened, indistinguishable, lardy mass. Microscopically, this consists of a finely granular substance in which there are here and there fine granular cells and the remainders of the nerve fibers. There appears to be scarcely anything left of the normal appearance of the latter. The majority show fatty degeneration; that is, they are filled with drops or with very small globules of fat, and in many places the myelin sheaths have entirely vanished, and only chainlike deposits of drops of fat remain to show that formerly nerves had been present here.

It can scarcely be doubted that constitutional syphilis has been the cause of these processes, especially when one compares this case with a similar one which von Graefe has reported in his *Archiv für Ophthalmologie,* in which there was present an irregular degeneration of the oculomotorius, even though apparently in a later stage of the process.

It cannot be denied that in this case also the nerve substance would have shown a similar atrophy as that mentioned before if the exudate which pervaded the affected trunk had gone on to resorption. It is interesting that in both cases there were areas of softening in the brain. In our case this softening was apparently the result of a plugging of the arteria profunda cerebri. How this occurred, whether by an embolus or through narrowing at the site, cannot be discovered. The heart showed no abnormalities, but in the large vessels there were atheromatous deposits here and there. Apparently this plugging occurred in 1849, on a night when the patient was awakened with apoplectic symptoms. The scarring

contraction of the parenchyma of the liver is also known to be characteristic of constitutional syphilis.

The similarity from the beginning of this case to dementia paralytica, which was further augmented by the grandiose delusions, the widening of the pupils and the chronic arachnitis, allows us again to bring up the question whether syphilis was not the cause of this last illness. In nine subsequently observed cases (with the exception of only a single one in which no visits to brothels could be determined) syphilis has been shown to be present. In none was hereditary syphilis present, which, according to French writers, is found to the amount of one third or one half of the total number of cases. According to the same authors (Bibliothèque du médecin-practicien 1x, p. 541), the number of paralytic men was four times that of women, which relationship corresponds rather well to the general prevalence of syphilis. In women of better social position dementia paralytica is scarcely ever present. In large cities it is strikingly common. Disorderly living and misuse of alcoholic liquors are contributory factors for that as well as for other diseases. These facts support the hypothesis that syphilis can be the basis for dementia paralytica. Whether this really is correct or not must be determined by further observations. The treatment of dementia paralytica by antisyphilitic remedies cannot be expected to give very good results, since the disease has reached the stage which causes the patient to be sent to an asylum. At least we have obtained no results in two cases by the use of potassium iodide or the sublimate. But in the beginning of the disease it may be otherwise, and attempts along this line would be of the greatest importance. In no case can the lack of success of the therapy in advanced disease be considered a partial proof against the correctness of the aforementioned hypothesis.

C. was an officer, 48 years old, with no hereditary taint. The patient had always led a very regular life. During the last twenty years and before his marriage in late life coitus was apparently practiced very little. He did, however, contract a primary syphilitic lesion, as the result of which he was upset to a very serious degree. For about three years he has suffered from gastric distress, a yellowish color of the skin, loss of weight and poor spirits, the latter alternating from time to time with minor elations during repeated "cures" in Kissingen. For a few months there had been present a gradually growing excitement, which progressed to an excessive feeling of self-complacency, delusions and a confusion of ideas and finally to a mania.

From its beginning on July 19, 1855, the mania developed quickly to the greatest frenzy, with all its known symptoms, and continuous tubs, morphia and laxative treatments were used. At the same time there developed over the whole body and especially on the legs an exanthem which showed almost immediately the characteristic symptoms of syphilis of the skin. The original form of this condition could not be exactly observed because of the unfavorable condition of the patient. Apparently there were large blebs (*Rhypia*) which were immediately scratched off by the patient and bathed in urine. Restraint was impossible because of the patient's tendency toward being easily abraded. There were round, red or livid spots of different size (even as large as a two "Thaler" piece) which were partly excoriated and partly covered with dried blood or with a thick brown scab (Rupia). When the latter fell off, copper-colored spots remained which later became brown and left behind here and there wrinkled scars.

The patient became worse toward the end of August. He was without appetite, had much spontaneous vomiting and was continuously sleepless, but showed no fever. Since the use of medicine was practically impossible, the treatment was

limited to the administration of a selected strengthening diet of wine and later of Bavarian beer. Toward the end of September the patient began to improve; he became stronger; sleep returned, and he felt enervated. He remained in bed and allowed the excoriated places to be treated with compresses of warm water, whereon these began to heal. The confusion continued, and toward the middle of November a marked excitement returned. Shortly afterward there appeared swelling of the knee joint, with a fluid exudate which distended the upper part of the capsule, the so-called bursa extensorum. Pressure on the fluctuating swelling above the patella allowed this to be pushed forward, and it could be thrust against the condyle of the femur with a definite sound. At first leeches and ice-bags were used; later, pressure bandages were applied which the patient immediately tore off. In December the swelling appeared to go down, but it increased later in the course of months. The excitement became more intense, and small pustules recurred in different portions of the skin. These the patient infected by scratching, and specific treatment had to be resorted to. This was carried out rather regularly but not without difficulty. The fact that the excitement was decreasing and interspersed with quieter spells, as well as the patient's own worry over the trouble in the knee, made the treatment with medicine more easy. From December 14 to 30, he received 2 ounces of ammonium iodide and 6 ounces of potassium iodide; then, since there was no improvement, he received 12 grains of sublimate from January 4 to 21, and then potassium iodide again in small doses because of the severe salivation. During the treatment with mercury the excitement increased, but not the confusion. The swelling of the knee diminished, and the small lesions in the skin, which were most prevalent at the corner of the mouth, began to heal. With prolonged use of iodides the psychic symptoms gradually improved, and the body became visibly stronger. In March the serous fluid in the knee joint was decreased by means of repeated use of plasters. In April, the patient was dismissed uncured; he promised to communicate to us the later developments of the disease process which we will then publish.

In this case, as in the first, the symptoms were related to a previous syphilitic infection, and this disease, which had not been admitted earlier by the patient, had later been made certain by questions of the physicians. We cannot doubt that in the last case syphilis was the cause of the disease, since the specific remedies again acted in an extraordinary manner equally on the body and on the mind. Should, however, the confusion which was strikingly unimproved remain, that would still be in accord with usual experience in which partial or total mental deterioration frequently remains after the disappearance of the primary somatic and psychic disturbance. Naturally three cases cannot determine the difficult question whether this is cause or complication, and it must be admitted that syphilis can also occur as a chance complication of a psychic disturbance, since mentally diseased patients can become infected and later have constitutional syphilis. We wished, however, only to express our belief concerning the aforementioned few cases and to stimulate similar observations and also differences of opinion.

SUMMARY

Three important early contributions to the history of neurosyphilis are presented. The first consists of the autopsies reported by John Haslam, in 1798; that work is considered to be the first published description of dementia paralytica.

Bayle's thesis (1822) on chronic arachnitis, considered as a cause of mental disease, is translated into English for the first time. In his

thesis Bayle reported six cases, complete with clinical history and observations at autopsy. These cases were examples that Bayle had separated out of the general run of mental disorders as a group that could be recognized both clinically and pathologically as instances of dementia paralytica.

The first published account of the etiologic relationship between syphilis and mental disease is credited to Esmarch and Jessen (1857), whose treatise, translated into English,[7] is presented with a comment on their conclusions.

These three contributions represent the source material that has been cited in medical literature for over a century; as the original articles are difficult to obtain they are given here *in extenso*.

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7. Mr. Joseph Sasserno of the Roxbury Latin School read and corrected the translation of Bayle's work, and Dr. and Mrs. Charles H. Kimberly of Stockbridge, Mass., assisted in the translation of the article by Esmarch and Jessen.

News and Comment

SECOND INTERNATIONAL NEUROLOGICAL CONGRESS

The Second International Neurological Congress will be held in London, England, from July 29 to Aug. 2, 1935. Scientific sessions will be conducted each morning and afternoon except Wednesday, July 31, which day will be devoted to excursions and nonprofessional activities. Four topics have been chosen for discussion and stated programs are being prepared as follows: (1) "The Epilepsies; Their Etiology, Pathogenesis and Treatment," chairmen. Prof. Otto Marburg and Prof. Ottorino Rossi; (2) "The Physiology and Pathology of the Cerebrospinal Fluid," chairman, Prof. Otfrid Foerster; (3) "The Functions of the Frontal Lobe," chairman, Prof. Henri Claude; (4) "The Hypothalamus and the Cerebral Representation of the Autonomic System," chairman, Prof. Bernardus Brouwer. Two sessions will be devoted to consideration of the epilepsies. The stated subjects will be discussed at the morning sessions. After the presentation of each stated topic and the contributions of invited discussers, the session will be thrown open to general discussion, each discusser being allowed five minutes for his remarks. The afternoon sessions will be devoted to the discussion of miscellaneous topics. A sufficient number of separate meetings will be arranged to take care of all contributions accepted by the program committee. The presentation of all papers and the discussion of the fixed topics and miscellaneous papers will be restricted to members of the congress.

Any recognized neurologist or psychiatrist may submit titles for presentation at the miscellaneous sessions. Such titles, accompanied by a brief abstract of not more than two hundred words, must be submitted to the United States committee before Jan. 15, 1935. Titles and abstracts should be sent to the secretary of the United States committee. Abstracts must be written in English, French or German. These titles and abstracts will be considered by the United States committee and the proposer notified of its decision. The titles and abstracts accepted will be forwarded to the secretary-general in London for final consideration, acceptance and placement on the program, or for rejection. The proposers will be notified of the action taken at the earliest possible date. Not more than one contribution will be accepted for presentation from any member of the congress unless he is one of the invited contributors to the stated programs, under which condition he will be allowed to present one additional paper at one of the sessions devoted to miscellaneous topics. So far as is possible contributions to be presented before the miscellaneous sessions will be arranged in appropriate groups. The maximum time allowed for the presentation of a paper at one of the miscellaneous sessions is ten minutes. There will be open discussion of the papers presented at the sessions devoted to miscellaneous topics, five minutes being allowed to each discusser. The discussion of any particular paper may be closed by the chairman if the exigencies of time render this necessary.

Candidates for membership in the congress may apply in one of two ways: either (1) through the national committee, or (2) by direct application to the secretary-general, Dr. S. A. Kinnier Wilson, 14 Harley Street, London, England. All candidates from the United States are urged to follow the first method. Application blanks may be obtained from the secretary of the United States committee. Candidates may apply for active or associate membership. Active members are physicians possessing the necessary ethical and professional requirements of physicians in good standing and of known attainments in neurology, psychiatry and allied branches of these specialties and also physicians and surgeons of recognized standing. The membership fee is the equivalent of 12½ Swiss francs, payable on receipt of the certificate of registration.

Thomas Cook and Son are the official travel agents for the congress. Members and their families intending to attend the congress should make arrangements for traveling and hotel accommodations through Thomas Cook and Son, who will function in close cooperation with the local British committee in charge of local details. Individuals may, of course, make independent traveling and hotel arrangements. In all probability a definite steamer arriving in England shortly before the beginning of the meeting will be chosen so that as many as so desire may travel on the same steamer. Definite announcement will be made later in regard to this and also any other details which may be of interest or importance.

Requests for application blanks for membership and the submission of titles should be made as soon as is practicable.

The committee from the United States includes: Prof. B. Sachs, chairman; Prof. Henry Alsop Riley, secretary; Prof. Harvey Cushing; Prof. Charles L. Dana; Prof. Adolf Meyer, and Prof. Frederick Tilney.

Obituary

THEODORE H. WEISENBURG

1876–1934

The untimely loss of Theodore H. Weisenburg, for many years editor-in-chief of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, will long be felt wherever there is an active interest in neurology. After a protracted illness, against which he struggled courageously, Dr. Weisenburg died in Philadelphia on Aug. 3, 1934. Most of his life was spent—and devotedly spent—in this center of American neurology. He was one of the younger members of the famous Philadelphia school, and by his own achievements added much to the luster of its world-wide renown.

Dr. Weisenburg was born in New York on April 10, 1876. He received his preliminary education in public and private schools, and in 1899 the University of Pennsylvania conferred on him the degree of Doctor of Medicine. On Jan. 1, 1901, after completing internships in the Lackawanna Hospital, Scranton, and the Philadelphia General Hospital, he entered the United States Army and served as acting assistant surgeon in the Philippines until Nov. 1, 1902. For the next eighteen months he worked as a general practitioner in Philadelphia under the guidance of Dr. J. W. McConnell, and then became associated with the staff of Blockley Hospital.

On July 4, 1909, Dr. Weisenburg married Miss Constance V. Field, whose father was for many years a practicing physician in New York.

Many subtle qualities distinguish the life of useful achievement. Not the least of these are constancy of purpose, unflagging enthusiasm, courage, clear vision, well balanced restraint and, above all, belief in the rightness of a cause, whether it is large or small—unchanging faith that the chosen course of life, whatever its fruits, shall not be altogether in vain for the world and day in which one lives. Still more elusive is that spiritual quality by which one impresses others and is impressed by them. Many names have been given to it. But what matter the name of that which is so far beyond the feeble reach of naming? Where would this harried existence be without it—how much better with more of it? This quality and a deep earnestness of purpose were predominant in Theodore Weisenburg's life. Those who sought his aid as a physician felt the uplift of his quick human understanding. His colleagues recognized his keen, appreciative interest in their efforts and found in him an unfailing source of encouragement. Yet it was not alone through his generous appreciation of others that he exerted

his stimulating influence. A fine critical sense, always considerate in expression and constructive in intention, made him welcome in counsel and as a friend. Added to these naturally inspiring gifts, another factor gave his influence its full dynamic force. He was an indefatigable worker. From the earliest days of his medical career, he was continuously absorbed in the solution of some challenging problem; to the last he devoted himself to his investigations with unremitting perseverance, and this makes the sense of loss all the greater, that a spirit so indomitable, with its possibilities still unfulfilled, should terminate its course in so brief a span of years.

Time and place offered a peculiarly fortunate setting for Dr. Weisenburg's medical life. American neurology had successfully passed through the early stages of its development. Great leaders, like Dr. Mitchell, had seen it safely launched and prosperously on its way. The work of the Philadelphia school had already gained international reputation. Such distinguished pioneers as S. Weir Mitchell, Charles K. Mills, William G. Spiller, Francis X. Dercum, Charles W. Burr and Wharton Sinkler were firmly established as recognized authorities in this field of medicine. Each member of this illustrious group had already become, as he will ever remain, a guiding star in the destinies of neurology. It was into this group of neurologic leaders that Dr. Weisenburg came as a young man. They were quick to recognize his capacities and worth. For many years he was privileged to work side by side with them, drawing enthusiasm from their ardent devotion to medicine and guidance from their wisdom. He profited from Dr. Spiller's scholarly mind with its vast fund of knowledge of pathology. He was impressed by Dr. Dercum's clinical and philosophical studies. Dr. Burr opened his eyes to the increasing need of better psychiatric understanding and training for the neurologist. As time passed, he became more intimately associated with Dr. Mills. This intimacy ripened into close friendship, from which he derived those intangible benefits born of association with him who has been called the Nestor of neurology in this country. Soon he found himself no longer a humble co-worker, but moving forward shoulder to shoulder with the others, one of the outstanding members of this noted group.

Dr. Weisenburg's investigative interests were largely in the field of organic neurology. That phase of neurologic study, to which his colleagues in Philadelphia devoted such productive efforts, quite naturally made the strongest appeal to him. The wealth of clinical material in the wards of Blockley Hospital, where much of his time was spent, afforded abundant opportunity for investigations of this kind. In consequence, the range of his clinical and pathologic studies was broad. Certain major themes, however, commanded his attention almost from

the first. His earliest contributions were made in collaboration with Dr. Mills and Dr. Spiller, and such collaboration, especially with Dr. Mills, continued for many years. In 1905, he published an illuminating paper on pseudobulbar palsy, with a report of three cases with necropsy

THEODORE H. WEISENBURG, M.D.
1876–1934

and three without necropsy. This early effort revealed all the promising handicraft of the skilled workman. Shortly afterward, he made his first contributions on two of his major interests, the pathology of cerebellar tumors and word blindness, with some discussion of the treatment of visual aphasia. Both of these subjects, in their broader implications,

were to remain uppermost in his thinking and research. The problem of the cerebellum, especially the discrete localization of functional control within this organ, engaged much of his effort. His methods of approach were clinicopathologic and experimental. On the clinical side, his contributions concerned the diagnosis of tumors and other lesions of the cerebellopontile angle (1908), the results of bilateral involvement of the cochlear and vestibular divisions of the eighth nerve (1908), the diagnosis of tumors in the posterior fossa (1915), the diagnostic significance of the ear tests of Bárány (1915), the clinicopathologic interpretation of diseases of the cerebellum in general (1921) and also equilibration and the vestibular apparatus (1922). His contribution on cerebellar symptoms and cerebellar localization (1914), which was based on clinical and experimental evidence, defined his position in respect to the discrete localization of function in this organ. He maintained not only that such localization is demonstrable, but that the development of asynergia, whether truncal or appendicular, has definite localizing significance in diagnosis.

Another problem in which he had intense interest was aphasia. It requires an intrepid spirit indeed to grapple with the subtleties of the language function. Many have entered the lists only to find the present equipment of neurologic knowledge and understanding still insufficient to bring any convincing and wholly satisfactory decision. Dr. Weisenburg had both the necessary courage and the tenacity. He heard the first call to combat early in his career when he analyzed a perplexing case of word blindness due to a lesion in the right hemisphere in a right-handed man (1905). The fact that he lived in the inquisitive atmosphere of men who themselves were continuously prying into this knotty problem accounted for much of his persistent interest. Again in 1914, but this time somewhat tentatively, he ventured at it once more in his contribution, "Anarthria and Its Relations to Aphasia." Finally, with fast ebbing strength, he devoted the last years of his life to a masterly attack on the problem, and barely lived to see it finished.

Although less militant in the doctrine of the specific localization of function than his frequent collaborator, Dr. Mills, he none the less had strong leanings in this direction. Much of his investigation was undertaken to gather evidence in support of this doctrine. The first results of these studies appeared in a contribution on the localization of the higher psychic functions, with special reference to the prefrontal lobe (1906), and was shortly followed by an analysis of conjugate deviation of the eyes and head with disorders of associated ocular movements in tumors and other lesions of the cerebrum (1906). Disturbances in somatic sensibility were matters of constant interest to him; in fact, the organization of a symposium on this subject was among his last

constructive activities. His contributions on astereognosis without sensory or motor involvement (1906), subdivision of the representation of cutaneous and muscular sensibility and of stereognosis in the cerebral cortex (1906) and sensory and motor disturbances in parts above the distribution involved by definite organic lesions of the spinal cord (1907) are valuable additions to this aspect of neurologic knowledge.

The localizing significance of lesions in the interbrain offered another field for investigation. Dr. Weisenburg's studies of a case of tumor destroying most of the optic thalamus and causing central pains, vasomotor and trophic disturbances, involuntary howling, and deviation of the body to the opposite side (1910) were incentives for further work on this region of the brain. Not a little of the extensive, present-day research devoted to the diencephalon was stimulated by his contribution on tumors of the third ventricle and the establishment of a symptom complex (1910).

During the widespread epidemic of poliomyelitis in 1916, the clinical material in Blockley and other hospitals gave him opportunity to survey and classify an exceptionally large number of patients. His classification of the clinical varieties of this disease (1917) has served as a guide in all subsequent studies of infantile paralysis.

Dr. Weisenburg's writings include numerous chapters in various systems of medicine. Among these are "Diseases of the Cerebellum" in Nelson's Loose Leaf Medicine; "Diseases of the Spinal Cord and Spinal Nerves" in the "Eye and Nervous System" by W. G. Spiller and W. C. Posey; "Diseases of the Nervous System" in "Surgical Diagnosis" by Edward Martin, and "Diseases of the Nervous System" in "Medical Diagnosis" by J. M. Anders and L. N. Boston. During the World War he compiled and edited the "Manual of Neurosurgery" of the Medical Department of the United States Army.

Even more stimulating, perhaps, than the results of his own investigations, was the influence which Dr. Weisenburg exerted throughout the entire country as editor-in-chief of the ARCHIVES OF NEUROLOGY AND PSYCHIATRY. At the close of the war, the trustees of the American Medical Association graciously offered to undertake the publication of a journal which should be, in part at least, the official organ of the American Neurological Association. Dr. Weisenburg was chosen editor-in-chief and retained this post until the time of his death. The first volume of the ARCHIVES appeared in 1919 and met with immediate success. Under his wise direction, this success continued to grow. The journal, which at first published 1,500 pages a year, now carries about 3,000 pages annually. Scientific contributions are received from all parts of the medical world, and the periodical has attained a circulation as large as that of any neurologic journal published in the English

language. Dr. Weisenburg organized an energetic and cooperative editorial board as well as a large staff of reviewers and abstractors. Most of the editorial work, however, fell on his shoulders. It was in this editorial capacity that many of his exceptional qualities found their fullest expression. He was continually on the look-out for promising workers in the field of neurology and equally intent on developing younger talent. His discriminating eye followed all of the modern neurologic trends, and his quick discernment of merit brought constructive recognition to many who might have fared ill without it. In this manner, particularly, his influence spread until it may justly be said that no single factor has had a greater effect in the growth and progress of neurology in this country than the publication of the ARCHIVES under the editorship of Theodore Weisenburg. This belief is shared by the associate members of the editorial board who came to regard him as an ideal editor-in-chief.

Dr. Weisenburg's activities were not wholly confined to the more sedentary pursuits of medicine. He had reason to be proud of his record as an adopted son of Mars. In addition to service in the Philippines, his services during the World War were many and varied. He was a member of the Executive Committee of the War Work Committee of the National Committee for Mental Hygiene in New York. Later he served for a year as contract surgeon in the United States Army Medical Corps and as director of the Military Neuropsychiatric Training School of the Philadelphia General Hospital. In 1918, he received the rank of major in the Medical Corps, acting as chief of clinic in General Hospital No. 30 at Plattsburg and subsequently chief of the neurological service in General Hospital No. 1 in New York. After the war he wrote the military history of the American Neurological Association, which appears in the association's semicentennial volume.

In addition to the demands of a large private practice, he gave much time to his service at the Orthopedic Hospital and Infirmary for Nervous Diseases from 1922 until his death. He was also on the staffs of the Philadelphia General Hospital, the Hospital of the Graduate School of Medicine of the University of Pennsylvania, the Municipal Hospital, the Norristown State Hospital and the Eastern Pennsylvania Institution for Feeble Minded and Epileptics at Spring City. He was also consulting neurologist at St. Joseph's Hospital and the Wills (Eye) Hospital of Philadelphia, the Homeopathic Hospital of Wilmington, Del., and the Chester County Hospital.

As a teacher, Dr. Weisenburg was clear and impressive. He had the same directness and simplicity of style that have distinguished the utterances of his Philadelphia colleagues. Much of his teaching was done in the postgraduate field. It was this interest that led to the pub-

lication in the ARCHIVES of the series of articles on neurologic and psychiatric education by leaders in different countries. For several years he was chairman of a committee on training in neurology of the American Neurological Association, and he was active in the establishment of the American Board of Psychiatry and Neurology. At the last meeting of the association he was elected chairman of its representatives on this board.

Dr. Weisenburg held positions as instructor in neurology and neuropathology at the University of Pennsylvania Medical School (1903-1907), professor of neurology at the Medico-Chirurgical College in Philadelphia (1907-1915) and professor of neurology in the Graduate School of Medicine of the University of Pennsylvania (1917-1934). He was among the first to utilize moving pictures in neurologic teaching and for the analysis of motor disturbances.

Admitted to the American Neurological Association in 1905, Dr. Weisenburg became vice president in 1917 and president in 1918. As editor of the transactions of the association, he served as a permanent member of its council since 1919. Success in the preliminary arrangements for the first Anglo-American Congress of Neurology held in London in 1927 and the International Neurological Congress held in Bern in 1931 was due, in no small measure, to his efforts. He was chairman of the Section on Nervous and Mental Diseases of the American Medical Association in 1908 and president of the Philadelphia Neurological Society. Since the founding of the Association for Research in Nervous and Mental Diseases, Dr. Weisenburg was active as a member of its commissions. In 1933, he was elected president of the association and had successfully completed the program for a symposium on sensation and sensory disturbances, to be presented at the annual meeting in December 1934. He was also a member of the Philadelphia Psychiatric Society, the Philadelphia College of Physicians, the American Psychiatric Association and the Association of Military Surgeons of the United States. In 1926 he was elected a corresponding member of the Verein für Psychiatrie und Neurologie of Vienna.

When Dr. Weisenburg was asked to give the first Charles K. Mills Lecture, he prized the honor highly. After more than four years of investigation, his Mills Lecture on aphasia was delivered and enthusiastically received in 1933 at Washington, D. C., during the sessions of the American Congress of Physicians and Surgeons. His conclusions were cautiously drawn. They had been carefully checked by a number of well devised controls. Broadly speaking, they went far toward dispelling the more extreme ideas of precise cortical localization for the language function. In fact, his major deductions "that language is the result of the unified activity of the whole brain" is categorically

opposed to earlier conceptions and more in accord with the trend of modern opinion. But even if certain vital questions in the problem of aphasia still remained open, it is probable that no clinical research more extensive and painstaking has yet been applied to this difficult subject. It was this aspect of the work that taxed Dr. Weisenburg's failing health and tested his courage. The vast amount of clinical observation had to be properly assembled, and the data correlated and made ready for publication in book form. Dr. Weisenburg recognized the magnitude of the task, little realizing that he was putting his scant energy into his swan song. With a characteristic persistency that defied the interruptions of illness and distress, he persevered. At length it was finished. The toil of all these months was over. Little remained to be done. He was able to gather the remnants of his strength sufficiently to visit New York with his manuscript and make arrangements for its publication. His book on aphasia was an accomplished fact. But the journey was an exhausting one because the vital spark was almost spent. And having reached only so far as the midafternoon of life—too early for all his earnestness and enthusiasm and influence—he ceased from his labors. He rests among the peaceful hills of Valley Forge—symbol of the unquenchable determination of the true American spirit. There is only a modicum of solace in this thought, but for the friend who may not see him again he still lives in happy memories.

FREDERICK TILNEY, M.D.

Abstracts from Current Literature

ARGYLL ROBERTSON PUPIL. LEO F. MCANDREWS, Arch. Ophth. **10**:520 (Oct.) 1933.

A discussion of the Argyll Robertson pupil is, from an ophthalmologic standpoint, always of interest. Argyll Robertson, in his first paper, emphasized four points: miosis, the effect of atropine and physostigmine (eserine) on the iris, color blindness and the condition of the retinal vessels. In his second paper, four cases due to disease of the spinal cord were reported. In one, the characters of tabes were well marked; in two, the form of spinal involvement was doubtful, while in the fourth, the symptoms of spinal disease were not well marked.

According to Wilbrand and Saenger, a true Argyll Robertson pupil must have the following characteristics: There is loss of the direct and indirect light reactions. The convergence reaction is increased and sustained. The pupillary diameter is less than 3 mm. The size of the pupil does not vary from time to time. The psychic and sensory pupillary play is lost or lessened. According to McAndrews, many other writers on the subject do not subscribe to this definition of a true Argyll Robertson pupil, the question of miosis usually being the point of disagreement. He thinks that miosis is an important essential, because on it depends the decision as to whether one is dealing with a true (syphilitic) or a false (non-syphilitic) Argyll Robertson pupil.

Three important theories are outstanding as to the site of the lesion, namely, that it is located in the spinal cord, in the ciliary ganglion or in the mesencephalon or aqueduct of Sylvius. A review of the literature shows much conflicting evidence. Pathologic research has given little help on the subject. At present, only a few investigators hold that the lesion is in the cervical portion of the spinal cord. A few French investigators think that the ciliary ganglion is the site. The majority of writers state that the site cannot be positively localized. From McAndrews' analysis of the various reports, it seems that the weight of evidence indicates that the lesion is in the vicinity of the oculomotor nuclei. The fact that the extrinsic muscles are often involved at the same time makes this supposition more plausible. The theory of Behr, that the lesion is in the connecting neuron which runs from the afferent pupillomotor fibers to the sphincter nucleus, is accepted by the majority of students and writers on this subject. Investigators who thought that lesions of the spinal cord accounted for the major number of cases did not examine the brain, especially to see whether the floor of the third ventricle was involved. Investigators who worked with the ciliary ganglion as its etiologic factor presented many conflicting opinions. For instance, Wilbrand and Saenger failed to support Marina's findings, while McGrath supported them. Redslob agreed with some of the findings of Dupuy-Dutemps and disagreed with others. Sala found positive neuritic changes in the short ciliary nerves, while Samaja found none. Such isolated incidents are included in this abstract, in brief, only to illustrate some of the many conflicting opinions.

The reports from investigators working with the mesencephalon are numerous and are much better worked out. There are perhaps fewer differences of opinion in this field, but Wilbrand and Saenger reject Spiegel's theory, while Spiegel in turn pointed out errors in the observations of other investigators and advanced some new hypotheses himself.

The fact that an occasional unilateral true Argyll Robertson pupil exists eliminates certain other difficulties; especially does it throw out the possibility of localization of the lesion in the afferent side of the pupillary reflex arc, because such a lesion would cause bilateral miosis, and in the afferent pathway, since in this path light and convergence fibers lie together.

Spiller discussed the Argyll Robertson phenomenon and maintained that it was a source of error to hold that the light reflex must be wholly abolished, and that the abortive Argyll Robertson pupil has much the same clinical value as the completely developed phenomenon. He believed that there must be a center in the oculomotor nucleus. Such a center is supposed to be in the Edinger-Westphal group.

Concerning the nonsyphilitic form of Argyll Robertson pupil there is almost as much confusion. Moore expressed the belief that this type of pupil must have certain characteristics. The light reflex is absent; the convergence reaction is often delayed; it is uniocular; it dilates fully in response to mydriatics; the patient shows no evidence of syphilis, and the pupil is not usually contracted.

From McAndrews' comment and from his review it seems that one can safely say that a real Argyll Robertson pupil is always a sign of syphilis until it is proved otherwise. If one holds that the true Argyll Robertson pupil has certain definite characteristics besides the loss of the light reaction, many cases must be excluded because they do not come under the classification of Wilbrand and Saenger. In many reported cases the pupils are dilated and the convergence reaction is retarded. Most writers on this subject hold that a real Argyll Robertson pupil has certain definite features in addition to the absence of the light reflex; they agree with Bumke and Behr that such a pupil is always a sign of syphilis of the central nervous system.

SPAETH, Philadelphia.

MENTAL EFFORT AND METABOLISM, J. A. M. A. **102**:540 (Feb. 17) 1934.

In "The Elements of the Science of Nutrition," Graham Lusk stated that the source of mechanical work must be the metabolism, for mechanical energy cannot be derived from nothing. The necessary energy might be obtained in one of two ways: either at the expense of a proportionate reduction in the quantity of heat liberated by the resting organism or by an increase in the amount of the metabolism. In the former case, work would diminish the production of heat and might cool the tissues, which is not observed to take place. If work were done at the expense of increased metabolism, and if this increase were completely converted into mechanical effect, the production of heat in the organism might remain the same as in the resting state. If, however, the result of mechanical effort should be a stimulation of metabolism to the extent of not only enabling the body to do work but also causing it to produce more heat than when at rest, the tendency of the tissues would be to grow warmer, perhaps with a resulting outbreak of sweat to reduce the body temperature through physical regulation. The last named is the actual process.

The fact that under conditions of muscular exercise an augmented metabolism ensues is consonant with the early discovery of Lavoisier indicating an increased uptake of oxygen when work is done. What is to be said about the effects of mental effort? The influence of heightened mental activity on metabolism has long been debated. Muscular contraction, the fundamental feature of mechanical work by the organism, is attended with evidences of chemical changes. The output of carbon dioxide is increased; the intake of oxygen must sooner or later be augmented; the production of heat is an inevitable by-product. For many years it was impossible to demonstrate conclusively that such metabolic alterations attend the function of at least certain parts of the nervous system. With the development of more delicate methods of measurement, however, evidences of small changes indicative of metabolism seemed to be obtainable when peripheral nerves were provoked to manifest their characteristic functions. The nervous structures constitute only a small part of the "active" tissues of the body; hence it is quite conceivable that relatively small chemical transformations would escape detection in estimations of the metabolism of the body as a whole.

The situation is well summarized in studies of the Benedicts (Mental Effort in Relation to Gaseous Exchange, Heart Rate and Mechanics of Respiration, Washington, D. C., Carnegie Institution, 1933, publ. 446) in the Boston Nutri-

tion Laboratory of the Carnegie Institution of Washington on the metabolism during special types of mental effort by university trained subjects. The mental effort, continued for four consecutive fifteen minute periods, consisted chiefly in the multiplication of pairs of two digit figures. Before and following the mental effort, measurements of metabolism were made with the subjects in complete mental repose or during "attention," the attention consisting of closing an electric contact whenever a white or a red light appeared in the field of vision. The metabolism was the same during repose and attention. Sustained, intense mental effort for one hour caused an increase in heart rate, a hardly measurable increase in respiratory rate, a marked alteration in the character of the respiration, a considerable increase in the apparent total ventilation of the lungs, a small increase in the exhalation of carbon dioxide, a smaller increase (on the average, 4 per cent) in the consumption of oxygen and the production of heat and a slight increase in the apparent respiratory quotient. In the periods of repose following mental effort, all the factors measured, except in a few instances of physical discomfort or fatigue, were lower than during mental effort and tended to return to the original levels prevailing before the mental effort. The results of the second series of determinations during mental effort on the same experimental day duplicated for the most part those of the first. There was no indication of a summation effect or of a greater increase in the second series. Furthermore, during the progress of the four consecutive fifteen minute periods of mental effort there was no evidence of any greater effect on the factors measured in the later periods. Measurements of skin temperature on the forehead indicated that there was no appreciable alteration in the blood supply to the skin of the head. No change in the insensible perspiration during mental effort was noted. According to the Boston physiologists, the small increases in the consumption of oxygen and the production of heat are in large part to be accounted for by the increased muscular activity accompanying the increased ventilation of the lungs and the increased heart rate.

On the basis of all these carefully established observations, one may well agree with the conclusion reached by the Benedicts that mental effort per se is without significant influence on the energy metabolism. Mental achievement, therefore, need not be curtailed because of the high cost of living—at least so far as calories are concerned.

EDITOR'S ABSTRACT.

COMPARATIVE ANATOMY OF THE MESIAL GENICULATE BODY. T. HORNET, Arb. a. d. Neurol. Inst. a. d. Wien. Univ. **35**:76, 1933.

The mesial geniculate body is relatively better developed in apes than in man. This is partly due to the smaller size of the thalamic nuclei in the former. Carnivora, particularly the dog, have a much better developed mesial geniculate body than man and the apes. In the former, this structure is in the lateral portion of the mesencephalon, in the oral portion of which it forms a definite protuberance extending toward the midline of the brain stem. The structure is poorly developed in rodents, in which it does not form an external protuberance of the mesencephalon. In Proboscidea (only the Indian elephant was studied) the mesial geniculate body is free posteriorly; orally, it is connected with the mesencephalon; it consists of two nuclei (dorsal and ventral) separated from each other by myelin fibers, and the pulvinar seems to be connected with the mesial geniculate body. The structure is extremely well developed, especially its oral and dorsal portions, in Artiodactyla.

The largest surface, relative as well as absolute, in frontal sections of the mesial geniculate body was observed in Natantia. This is of interest because it is generally accepted that aquatic mammals do not possess good hearing; it is more than likely, therefore, that this unusually well developed structure in these animals may be the center for equilibrium.

The mesial geniculate body shows the greatest variations in size in Marsupialia. Whereas it is moderately developed in the kangaroo, Phascolarctus, Parameles,

Didelphis and Dasyurus, it is unusually well developed in Phalangista vulpina; in frontal sections it occupies one fourth of the surface of the mesencephalon.

The classic mesial geniculate body, though connected with various adjacent structures, consists of two principal nuclei. In man and in apes the ventral nucleus is situated at the posterior pole of the structure under discussion and disappears at the beginning of its oral half, and the dorsal nucleus occupies the oral pole and does not reach the caudal end of the structure. The dorsal nucleus is often subdivided in its oral portion by three lamellae. Between these two nuclei there is an important bundle of myelin fibers which originate from the side of the mesial geniculate body or possibly, as Cajal believes, from the lateral lemniscus. The dorsal nucleus is also traversed by a bundle of fibers originating laterally to the anterior quadrigeminal body and also perhaps from the lateral lemniscus. The division into two nuclei is less definite in Chynopithecini and in the lower apes. In spite of the greater development of the mesial geniculate body in the Prosimii, the division into two nuclei is not very distinct. In Carnivora, three groups of cells can be recognized, the middle one of which tends to spread laterally toward the inner portion of the mesencephalon. These three groups are not sufficiently distinct to be considered as separate nuclei. Rodentia, Proboscidea and Artiodactyla show a definite division into three or four nuclei. In Natantia, there is no subdivision of the mesial geniculate body. In Marsupialia, it is divided into four nuclei (dorsal, ventral, mesial and lateral), and in Phalangista, in spite of the enormous development of the structure, there are only two nuclei in it.

Under the designation of corpus parageniculatum mediale, Hornet describes a mass of gray matter attached to the mesial and oral portion of the mesial geniculate body. In myelin sheath preparations the corpus parageniculatum mediale appears to be continuous with the mesial geniculate body; with the Nissl stain both these structures are seen to consist of the same type of cells. The mesial parageniculate body is well developed in Carnivora and less so in Rodentia, in which it is made up of a mesial and an inner nucleus. In Marsupialia, Artiodactyla, Phocaena and Phalangista vulpina, the structure is situated close to the midline of the mesencephalon, although it is not distinct and is not subdivided. Hornet believes that this structure is analogous to the ganglion accessorium of the mesial geniculate body described by Marburg in man.

Von Monakow has designated a portion of the posterior nucleus of the pulvinar which is intercalated between the mesial and the lateral geniculate body as the ganglion intergeniculatum. In large apes this nucleus is a distinct structure which is surrounded by a dense capsule. In most animals, fibers from the lemniscus lateralis can be traced into the pulvinar and the mesial geniculate body as well as into the intergeniculate body. It appears from this that, in some respects at least, the functions of the intergeniculate body are similar to those of the structures with which it is connected. The relationship of the intergeniculate ganglion to the mesial geniculate body becomes clear when its position and structure are investigated in the lower mammals. The intergeniculate ganglion can be distinguished in Chirogaleus as that part of the pulvinar which appears in caudal sections dorsal to the middle geniculate body, constituting an integral part of it. In most of the other mammals the association of the intergeniculate ganglion with the mesial geniculate body is still closer.

KESCHNER, New York.

SOME NEW OBSERVATIONS ON THE DIAGNOSIS AND TREATMENT OF SYPHILIS. THOMAS ANWYL-DAVIES, Brit. M. J. **2**:487 (Sept. 9) 1933.

Primary syphilis may cause symptoms before it has reached the stage when secondary signs are apparent. Anemia, pains in the joints, persistent headaches, nausea and pyrexia occur in about 50 per cent of primary cases. Absence of inguinal lymphadenitis does not enable one to dismiss the possibility of syphilis in differential diagnosis. In women, in 44 per cent of cases the primary lesion appears on the cervix, and "the cervical chancre, hidden and painless, is the greatest

cause of the spread of syphilis." Points in differential diagnosis between a cervical chancre and other lesions are given.

A new treatment schedule is reported. It was based on the following observation: Spirochetes were studied for days by the dark-ground method. It was found that those bathed in arsphenamized serum gradually lost their normal appearance, became ghostlike, fragmented and paralyzed, and in about five days apparently died. Then, in five or six hours they recovered a normal appearance, became revivified, regained spirals and motility, and became as normal and as active as control spirochetes which had not been similarly "doped." This suggested that arsphenamine should be injected at intervals of five days or less, instead of waiting the customary week, which gives the spirochete three days in which to recover. The treatment schedule is: from the first to the fifth week, neoarsphenamine intravenously, 0.45 Gm. weekly, and bismuth 0.2 Gm.; from the sixth to the eighth week, potassium iodide, from 5 to 15 grains, three times daily; from the ninth to the thirteenth week, repeat as during the first five weeks, namely, total arsphenamine, 7.5 Gm., and total bismuth, 4 Gm., in thirteen weeks. The patient should report for a blood test in seven days after the last injection of the course. The patient should report again in twenty-eight days, and then be given potassium iodide until the next course begins. The weight of the patient is observed, and tests for albuminuria and urobilinogen are made at each injection. A prophylactic containing liver extract concentrate and dextrose is swallowed before each injection. Each patient is given three courses, with an interval of rest of eight weeks between the courses. Intravenous injections or deep subcutaneous injections once a week only, spaced as already outlined, may be given to weakly patients—especially to those over 45 years of age, or those with late syphilis.

Persons over 40 years of age eliminate drugs more slowly, and the course recommended is therefore not suitable for elderly patients. In early syphilis, during the acute infectious stage, the dose in the first week should be the strongest that the body can stand with safety. This same dose should then be maintained but not increased each week. For the average patient this means from 0.6 to 0.75 Gm. of neoarsphenamine a week. The deep subcutaneous method is therapeutically more efficient and safer, and causes fewer toxic complications than the intravenous.

The effects of treatment on the Wassermann reaction in 241 consecutive cases in which the full course was completed showed that in 1.24 per cent the reaction was positive, in 0.82 per cent doubtful and in 97.9 per cent negative.

"It is now the fashion, for fear of arsenical complications, to lessen the amount of arsenic, and to rely more on bismuth, which is far below arsphenamine in efficiency. This, and the lessened use of mercury, is, I think, a backward step. Two or three random injections of arsphenamine are inadequate, and destroy the patient's natural immunity."

The pregnancies of 300 syphilitic mothers were analyzed before and after treatment. Before treatment these 300 mothers had had 739 pregnancies, of which over 50 per cent were unhealthy. After treatment the total number of pregnancies was 305, of which just over 9 per cent were unhealthy. The mothers who had had less than one course of injections—that is, less than 5 Gm. of neoarsphenamine—had 30 per cent unhealthy pregnancies, while those who had had more than one course of injections had only 3.75 per cent unhealthy pregnancies.

FERGUSON, Niagara Falls, N. Y.

TREATMENT OF POLIOMYELITIS: PREVENTION OF DEFORMITY AND PROTECTION OF MUSCLES. JOHN RUHRÄH, J. A. M. A. **100**:1587 (May 20) 1933.

The following principles for the management of poliomyelitis are given: 1. Deformity may be prevented. The patient should be kept recumbent in a suitable splint which will help relieve the acute pain so often present at the onset. The nature of the splint will depend on the location and extent of the paralysis, but the main point to be remembered is that man is an erect animal and that the two muscle groups especially developed to enable him to stand erect, so differ-

entiating him from the ordinary quadruped animals, are the glutei and the quadriceps. These muscles should therefore always receive especial attention, and they will be conserved best by keeping the patient, while recumbent, in the position he would be in if standing. A modified double Thomas frame splint made to extend from head to foot can be used to maintain the body in line so that eventually, when the splint is discarded, the patient will be able to stand erect, assuming that he will have recovered sufficient power to do so. 2. Relaxation and rest are the most important factors in treatment. Fatigue, both general and local, must be guarded against. 3. The muscles must be protected. Overstretching a normal muscle will paralyze it temporarily, and overstretching a paralyzed muscle may damage it permanently. A muscle will recover its power more quickly and completely in complete relaxation than in any other position. A relatively weak muscle may in this manner become stronger than the one which originally opposed it, and a change in splints must be made accordingly. 4. Contractures must be prevented. They are due to shortening in unopposed and relaxed muscles, tendons and fascia. Proper splinting and the daily bath, at which time proper passive exercises may be undertaken, will prevent them. 5. At first the more important muscles should be favored until their recovery is assured. These are the deltoids, the extensors of the wrist, the opponens of the thumb, the glutei, the quadriceps, the calf muscles, the invertors of the foot and the abdominal muscles. 6. Muscles may be reeducated and strengthened by suitable exercise. In the early stages a daily warm saline bath will be all that is needed. Later, when the pain and tenderness have passed off, a small amount of daily exercise may be given. 7. The exercise given must always be well within the capacity of the muscles. The exercise must not be by rule of thumb but should be regulated each day, for each patient and for each muscle group. 8. The treatment should be individual and modified from time to time to suit conditions as they arise. 9. The muscles should be protected and trained as long as there is any improvement. 10. When muscles of the trunk and legs are involved, weight bearing should not be allowed while improvement is going on if the aim is to achieve all the recovery possible, but only after it has come to a standstill. The time to let the patient up is always a question. Macnamara continues a regimen of accurate splinting in the recumbent position as long as there are signs of improvement. Prolonged treatment often avoids the use of braces later. 11. Warmth is useful. The muscle should be warmed before exercise. Dry heat may be applied in tents or boxes fitted with carbon bulbs, improved appliances or other means. Fresh air, sunlight and ultraviolet rays in properly graduated doses are of value in improving the general condition. Massage, if used, should be gentle and, if there is any tenderness, avoided. Warm baths are most useful and are gratifying to the patient. A warm bath, preferably of salt water, should be given daily. 12. The morale of the patient should be kept up. Suitable amusements for young and old, education by mail or by teachers, games, radio, music and visitors help to pass the hours. An unoccupied person cannot be expected to be happy. Education, diversion or visiting should not be overdone. Proper mental rest is as essential as physical rest. EDITOR'S ABSTRACT.

MODERN TREATMENT OF BRAIN ABSCESS. H. P. CAHILL, J. A. M. A. **102**:273 (Jan. 27) 1934.

Cahill states that success in the treatment of abscess of the brain rests on a triad of medical actions: a correct diagnosis, proper operative methods and rational after-treatment. In cases of acute frontal sinusitis with probable abscess of the frontal lobe, a vertical incision in the median line joining the horizontal Killian incision enables the scalp to be sutured back, exposing the frontal bone. The anterior bony wall of the frontal sinus is then removed. The posterior wall of the frontal sinus is always carefully examined for exposed dura. The posterior wall of the sinus and the adjoining portion of the frontal bone are then removed. In about 50 per cent of abscesses of the frontal lobe, diseased dura or a small dural fistula is present; the tip of a small, no. 14 catheter is gently inserted for drainage

and immediate relief from symptoms due to pressure. Either of two methods of after-treatment is now possible. The first method includes the dilation of the fistula from every forty-eight to seventy-two hours by the insertion of a suitable larger catheter until a no. 24 catheter drain is accepted. In abscess of the frontal lobe adjacent to chronic involvement of the frontal sinus, the removal of a cone of cerebral tissue reaching to the capsular wall by means of the radio knife and loop of Bovie has been the method employed. It permits the use of the larger size of the Mosher wire gauze drain. The drain can be placed more accurately with little trauma or hemorrhage. It is safer to encircle the exposed dura by diathermy. A crucial incision is made in the dura to ascertain whether a superficial abscess or flat arachnoid lake is present before exploration is done for a deeper abscess. When the wire drain is suitably placed, various aids, such as rotation of the head, gravity, lowering of the top of the table and suction, may be employed to assist the cerebral pressure in promoting drainage and obliterating the abscess cavity. The formation of a fistulous tract about the drain to the abscess cavity makes the capsular wall extend to the surface of the brain. The drain, if in proper position, is allowed to remain undisturbed for about four weeks. The sutures in the scalp are then removed, and the brain is permitted to expel the drain gradually. During this period of drainage, little trauma due to manipulation of the drain is transmitted to the walls of the abscess. In abscesses of the temporosphenoidal lobe, approach through a subtemporal decompression has grown greatly in favor. In moribund patients with a sclerotic mastoid, it is the best method. In the treatment of cerebellar abscess, as in abscess of the middle fossa, if symptoms or signs of labyrinthine involvement preceded those of cerebellar infection, the paramastoid route through Trautmann's triangle, with, at times, obliteration of the lateral sinus, must be considered. When the abscess is secondary to involvement of the sinus, drainage through the inner wall of the sinus has been the author's customary procedure. Examination of the inner wall of the sinus may demonstrate a possible lead or a thrombosed plug in a small venule. If exploration of the mastoid cells posterior to the sinus reveals the presence of an extradural abscess, this area of protective meningeal adhesions should be used. The ideal treatment of an abscess of the brain would be to remove it in toto. This has been successfully accomplished; but the surgical shock, the encephalitis, the hemorrhage and danger of infection from the stalk, if present, or from the diagnostic puncture or from rupture of the capsule are insurmountable obstacles. Partial removal of the wall of the abscess by unroofing it, with herniation, greatly decreases the danger of the removal in toto. Dandy's method of repeated tapping of the abscess relieves the excessive intracranial pressure for a time and hence furnishes the time factor necessary for proper capsulation of the abscess.

PSYCHOLOGIC CONSIDERATIONS IN ALCOHOLIC HALLUCINOSIS: CASTRATION AND DISMEMBERING MOTIVES. WALTER BROMBERG and PAUL SCHILDER, Internat. J. Psycho-Analysis **14**:206, 1933.

The material in this article deals largely with cases of acute alcoholic hallucinosis, because such cases are most common in American psychiatric practice. Delirium tremens is infrequent and usually secondary to trauma or infection. In the period from 1928 to 1930, the ratio of hallucinosis to delirium tremens at the Bellevue Hospital in New York was 4.05 to 1. In European clinics the ratio is the reverse; there are four times as many cases of delirium tremens as there are of hallucinosis. In ten cases of alcoholic hallucinosis and in one case of toxic psychosis due to drugs there were expressions of castration and dismembering motives in the foreground of the clinical picture. These motives were closely connected with homosexual and anal trends. Since circumstances did not allow psychoanalytic investigation, it is impossible to say whether these motives played an important part in the patient's psyche or whether they were created by the toxic influence of alcohol. However, from knowledge of the personality and psychosexual organization of persons with alcoholism, it is probable that these motives

express in the psychosis those psychosexual elements already present in the make-up of most of such persons. Many of the authors' patients showed signs of sexual maladjustment prior to alcoholism, although there was no manifest homosexuality. It is easy to reconstruct the complete content of the motives in the various psychoses. The male patients (ten of the eleven patients were men) feared that they would be castrated and be used afterward as women. In the manifest content of the psychosis the fear was based on the hallucinations and delusions. The persecutors (in the minds of the psychotic men) were chiefly other men—soldiers, drinking companions and similar associates—who undoubtedly had formed part of their environment previous to the development of the psychosis; their choice of these associations was influenced by latent homosexual tendencies. The castration complex in psychotic women may appear in a different way. They fear losing the body cavities by occlusion or by extirpation of the cavity organs such as the uterus. The castration motive in women expresses itself in the delusion of being cut to pieces or of being dismembered. The castration motive is built up at different levels. At the most primitive level, it is represented by the fear of losing the entire body. At a higher level, there is the fear of the loss of a part of the body, and on a still higher level, there is the fear of complete loss of the capacity for sexual sensation. The retrogression from a castration to a dismembering motive is aided by the fact that castration provides possibilities for passive homosexual satisfaction—a possibility which the person suffering from alcoholism cannot admit. The patient, through his feeling of guilt, transfers his fears from the sexual parts to various other parts of the body. The death threats and the ideas of persecution satisfy the patient's masochistic tendencies associated with the feeling of guilt. Suicidal attempts can be regarded as an expression of a tendency to self-castration and self-dismembering. The authors believe that castration and dismembering motives are basic in cases of alcoholic hallucinosis.

KASANIN, Howard, R. I.

A HYPERKINETIC DISORDER OF CHILDHOOD. F. KRAMER and H. POLLNOW, Monatschr. f. Psychiat. u. Neurol. **82**:1 (Feb.) 1932.

Kramer and Pollnow observed forty-five children who presented a syndrome that has not been clearly differentiated from other disorders. The outstanding feature was motor unrest. This usually began between the ages of 3 and 4 years though an earlier onset was not uncommon. There was constant restlessness, of a purposeless nature, with great distractibility. Aggressive and negativistic traits and outbursts of rage were frequently noted. All but three of the patients showed disturbances of speech. As a rule, articulation was indistinct and only a few single words or unintelligible sounds were uttered. On the other hand, understanding of speech often seemed normal. While systematic examinations were not possible, the impression was obtained that most of the patients were of subnormal intelligence. The authors believe that in many instances this impression was falsely created by the extreme excitment and the disability in the sphere of speech. Convulsive seizures occurred in nineteen cases. As a result of later observations made in fifteen cases, Kramer and Pollnow found that the illness ran a constant course. After the age of 6 the symptoms gradually diminished in severity. In most instances the motor unrest, the disturbances of speech and the convulsive seizures disappeared completely. However, a considerable number of the patients showed permanent impairment of intelligence. Earlier writers have apparently described similar cases, which they generally classified with erethistic forms of mental deficiency.

The fact that one of the patients had an identical twin who remained normal caused Kramer and Pollnow to suspect that the syndrome described by them owed its origin to some exogenous process. Histologic observations made in a case in which sudden death occurred tended to confirm this view. The brain stem showed evidence of a chronic inflammatory process. There were perivascular

infiltrations in the interbrain and basis mesencephali and a subependymal increase of glia fibers in the region of the third ventricle, aqueduct of Sylvius and fourth ventricle. The substantia nigra showed severe degeneration of the nerve cells, a marked increase of glia fibers and compound granular corpuscles in the perivascular spaces. The authors point out that the histologic picture resembles that in epidemic encephalitis. Clinically, too, the patients presented certain features which were reminiscent of postencephalitic behavior disorders. However, neurologic signs were entirely lacking and the motor unrest failed to interfere with sleep. Furthermore, in some instances the illness occurred many years before the first cases of epidemic encephalitis were described. Hence the authors believe that epidemic encephalitis can be excluded as the etiologic factor. While histologic observations in only one case are not sufficient to warrant final conclusions, the lack of variability in the clinical pictures strongly suggests that the syndrome owes its origin to some pathogenetically uniform disease. ROTHSCHILD, Foxborough, Mass.

TYPES OF MENTAL DEFICIENCY AND THEIR SOCIAL SIGNIFICANCE. E. O. LEWIS, J. Ment. Sc. **79**:298, 1933.

Mental deficiency is nothing but a variation from the normal in the range of mental equipment, and since the range of normal variation is not known one should not assume that a mental defect is necessarily an evidence of abnormality. Mental deficiency that is abnormal belongs to a definitely organic pathologic group, which presents structural disease and abnormalities of the central nervous system. These cases should be called pathologic types. The person with mental deficiency who simply varies in his intellectual equipment from his fellow man should be called a subcultural type. Lewis points out that the deficiency is only an extreme example of normal variation of mental endowment. There is no definite cleavage, but a close biologic kinship, between this group and the mass of normal persons. This is altogether different from the case of the so-called pathologic defective.

Such a classification as Lewis offers gives an opportunity to understand the social problems involved. The general conclusions about the subject at present are that most pathologic cases belong in the lower grades of deficiency, idiots or imbeciles, whereas most of the subcultural cases belong in the class of feebleminded or what would be called in American terminology, "morons." The great majority of defectives belong to the subcultural group. The pathologic cases are evenly distributed among all the sections of the community. The subcultural type is concentrated to a large extent in that part of the community which is usually assisted by social agencies and which presents all types of social maladjustment like pauperism and slumdom. (This generalization by Lewis is probably not generally accepted by American students.)

Heredity is a more important factor in the production of subcultural types than in the production of pathologic types. When one finds several defective persons in one family they are usually of the subcultural types. Lewis points out that under modern conditions there is a tendency for the subcultural groups to be precipitated from the general life of the community. Modern conditions effect the exclusion of men and women with poor mental equipment. (This impression equally does not apply to American practice; with high development of industry there is an extra opportunity for the mentally defective person to do simple routine, monotonous, repetitive tasks.) Lewis believes that the subcultural types constitute greater social problems than the pathologic types, because in cases of the pathologic type the only procedure available is institutional treatment, whereas subcultural types may be adjusted in the community. He believes also that the same principles should be applied for the purification of the community as in the case of tuberculosis; by this he probably means eugenic control. KASANIN, Howard, R. I.

MALIGNANT HYPERTENSION: REPORT OF CASE TREATED BY BILATERAL SECTION OF ANTERIOR SPINAL NERVE ROOTS FROM THE SIXTH THORACIC TO THE SECOND LUMBAR, INCLUSIVE. A. W. ADSON and G. E. BROWN, J. A. M. A. **102**:1115 (April 7) 1934.

Adson and Brown report the case of a patient who presented a severe, progressive form of essential hypertension. Organic changes in the retinal arterioles and active retinitis were present. One month following resection of the anterior spinal nerve roots there was a definite drop in blood pressure when the patient was recumbent, but this lowering of pressure was accentuated by the upright posture—relative hypotension. Restoration of this patient's diastolic blood pressure to normal levels was impossible because of the structural hypertrophy of the arterioles. While this patient is active and up and about, the range and variation of her blood pressure are those of a moderate degree of hypertension, and during this active period the overload on the heart and other organs is materially diminished. The argument that high levels of blood pressure of patients with hypertension should be maintained for the kidneys to function adequately is not demonstrated in this case. The activity of the heart and kidneys is well maintained, with a drop of 80 mm. of mercury in systolic pressure. The excretion of water was not materially modified but was merely delayed. A change in the site of the arterioles of the retina could not be demonstrated in this short period, but decrease in the activity of the retinitis was noted. The pressor response, after section of the anterior roots was comparable to the effects of psychic and emotional stimulation on the blood pressure. It constitutes an accurate measuring rod to determine the effects of treatment on the variability of the blood pressure. Following operation there was a significant decrease in the maximal rise in blood pressure. This was more marked when the patient was upright, namely, a decrease of 100 mm. of mercury in the response of the systolic pressure. This effect was further confirmed by the postoperative hourly readings of blood pressure taken under various forms of mental and physical activity. Denervation of the vessels below the diaphragm of the patient did not completely inhibit the pressor responses to cold; this may have been the effect of the vasoconstrictor response of the arteries of the upper part of the body. The effects of the operation on the sweat and vasomotor fibers indicated that almost complete degeneration of the sweat fibers had taken place below the level of the episternal notch. It is probable that the depressor effects of this operation were the result of two factors: (1) loss of vasomotor control of the splanchnic and peripheral arteries below the diaphragm, and (2) lowering of intra-abdominal pressure through paralysis of the abdominal muscles.

EDITOR'S ABSTRACT.

SUBARACHNOID INJECTIONS OF PROCAINE HYDROCHLORIDE: QUANTITATIVE EFFECTS OF CLINICAL DOSES ON SENSORY, SYMPATHETIC AND MOTOR NERVES. J. L. EMMETT, J. A. M. A., **102**:425 (Feb. 10) 1934.

Emmett endeavors to present graphically the sequence of physiologic events that occur in spinal anesthesia with a wide range of dosage and concentration. The study demonstrates the variable factors that determine the action of the anesthetic drug in individual cases and shows that small doses of procaine hydrochloride, not sufficient to produce anesthesia, seem adequate to establish complete vasodilatation. His series of thirty-four cases consists of persons of both sexes and of a wide variety of age groups. Thirty-one of these patients were subjected to spinal anesthesia with procaine hydrochloride, one to spinal anesthesia with pantocaine (*p*-*n*-butylaminobenzoyl-dimethylamino-ethanol hydrochloride), and two to general anesthesia by inhalation. Observations were made at short intervals on the amount of block produced in the sympathetic, sensory and motor nerves, with a wide range of doses and concentrations. The physiologic sequence of nerves blocked in spinal anesthesia was found to be, in order of appearance, (1) sensory nerves, (2) sympathetic nerves and (3) motor nerves. However, it is likely that the sympathetic nerves are actually the first to be blocked. The

means of measuring block of the sympathetic nerves is necessarily indirect, depending on the rise of surface temperature of the area supplied by the blocked nerves, and because of this, delay is recorded after block of the sensory nerves. The order of recovery is (1) motor nerves and (2) sensory and sympathetic nerves at about the same time. Fractional block is possible. Production of block in one system and not in another, by variation of dose and concentration, is feasible. Procaine, 60 mg., in a concentration of about 1.5 to 2 per cent is adequate to produce a rise in cutaneous temperature to a level assumed at the present time to indicate complete vasodilatation. Complete anesthesia is not necessary to secure this vasodilatation. Complete sensory effect is usually had in from four to eight minutes. For practical purposes an interval of thirty minutes after injection should be sufficient to produce the maximal rise in surface temperature due to block of the sympathetic vasoconstrictor nerves. The time between the injection and the production of block of the motor nerves is variable, ranging from three to twenty-three minutes. All these figures vary greatly according to the dosage and concentration used. EDITOR'S ABSTRACT.

THE PLACE OF PSYCHIATRY IN MEDICAL EDUCATION. RALPH A. NOBLE, Ment. Hyg. **17**:1 (Jan.) 1933.

The general practitioner tends to regard disorders of a mental nature as being quite outside the scope of his work. This is unfortunate, for there is no reason why the family doctor should not be able to deal adequately with the rank and file of human problems. One way to make it possible for him to do so is to see that every general hospital has an active psychiatric consultation service, and that the consultants are good doctors as well as good psychiatrists. The lack of cooperation and understanding will not be completely eradicated until the medical schools assign to psychiatry its proper place in the education of the physician. While the student is studying anatomy and physiology he should learn something of their psychic counterpart, psychobiology, as Meyer terms it. The importance of personality trends should be demonstrated, and this, not by a psychologist or by an internist but by a qualified psychiatrist. During the last two clinical years attention should be given to the teaching of the commonest types of mental reactions. The clinic for mentally ill persons must be in close association with the general hospital. Stress should be laid on the appreciation of the patient as an entity, and it is better that the student apprehend the general psychiatric aspect of his cases than that, ignorant of this, he be able to distinguish a manic from a schizophrenic patient.

The opportunity of giving practical psychiatric training during internship must not be overlooked. This, of course, is possible only when an active psychiatric consultation service is available. The agencies outside the hospital—clinics, child guidance departments, probation offices and social service bureaus—also offer valuable training in psychiatric approach, material and methods to the intern who is interested.

The thorough training of the psychiatrist is a postgraduate matter, and in the United States and Canada not more than five or six universities give postgraduate tuition in this specialty. Noble believes that before undertaking special study in this field, it is well for the graduate student to have had some years of general practice. By such methods, not only will the profession be enriched by better trained specialists, but the community will receive physicians who are able to safeguard more adequately its mental welfare as well as its physical health.

DAVIDSON, Newark, N. J.

ENCEPHALOGRAPHIC AND HEREDITARY INVESTIGATIONS IN IDIOPATHIC EPILEPSY. H. BOENING and T. KONSTANTINU, Arch. f. Psychiat. **100**:171 (July) 1933.

Boening and Konstantinu review the literature on idiopathic epilepsy with special reference to investigations concerning the etiology and pathology of the disease. They undertook an investigation of two aspects of the problem, namely, the search

for signs of organic brain disease as demonstrated by pathologic encephalographic findings and the search for indications of hereditary factors. They selected from a group of 295 persons with epilepsy 97 who undoubtedly had so-called idiopathic epilepsy. Their observations were as follows: Half of the patients showed normal encephalographic pictures of the ventricular system. About the same proportion showed a normal encephalographic picture of the superior surface of the brain. Some with pathologic findings, however, overlapped a certain proportion of the group in which the ventricular system was normal, and vice versa. It was found that persons who have suffered with the disease for a long time tend to show more encephalographic defects than those in whom the disease has been of short duration. This tends to indicate that the defects that are demonstrated by encephalography may be results rather than conditions associated with the cause of the disease. No relationship was found to exist between pathologic encephalographic findings and such clinical features as original feeblemindedness, epileptic deterioration or organic neurologic signs. In 4 cases convulsions were elicited by encephalography; no definite improvement was noticed in any case following the procedure.

The investigations into heredity showed the following results: Of the 420 siblings of the patients investigated, only 4.3 per cent suffered from epilepsy, 2 per cent were feebleminded and 6.4 per cent were left-handed; no cases of typical migraine were found in any of the siblings. Thirty-two of the patients had children (94); of these, none showed definite epilepsy, but 15 were feebleminded. Study of the parents of the patients showed true epilepsy in only 1 father (0.52 per cent of all the parents). Of 793 collaterals studied, only 9 showed definite epilepsy, while 6 were suspected and 1.06 per cent showed feeblemindedness. Of the 388 grandparents, only 2 showed definite epilepsy and 2 were suspected. The percentage of alcoholic addicts in all relatives was not appreciably different from that in the normal population.

MALAMUD, Iowa City.

EXPERIMENTS WITH ANESTHETICS: IV. LESIONS PRODUCED IN THE SPINAL CORD OF DOGS BY A DOSE OF PROCAINE HYDROCHLORIDE SUFFICIENT TO CAUSE PERMANENT AND FATAL PARALYSIS. JOHN L. LUNDY, H. E. ESSEX and J. W. KERNOHAN, J. A. M. A. **101**:1546 (Nov. 11) 1933.

The experiments of Lundy, Essex and Kernohan on dogs show rather conclusively that the concentration of procaine used in spinal anesthesia is quite as important as the total dose. Consequently, recommendations concerning the dose of anesthetic agent, in terms of body weight, should always be accompanied by recommendations concerning the concentration in which the agent should be given. The authors are unable to offer any satisfactory explanation for the permanent effects of certain doses of procaine in certain concentrations. It has been shown that the permanent changes in the cord that result in permanent paralysis of the hindlimbs of dogs given 5 cc. of from 20 to 25 per cent solution of procaine are not due to intradural pressure or to the osmotic pressure of the solution. It might be argued that the long period of low blood pressure that invariably accompanies such spinal anesthesia might result in anemia of the spinal cord, but in certain instances permanent effects did not follow complete respiratory paralysis that endured nearly three hours, during which time the authors have reason to believe the blood pressure remained at a level comparable to that of dogs permanently affected. The central nervous system of the dog is capable of withstanding the action of huge doses of procaine. The doses used for man are proportionately much less. It is probable, therefore, that many of the accidents following the use of spinal anesthesia in man are due to factors other than the action of the anesthetic agent. This may not be true of some other local anesthetics, since Spielmeyer, working with dogs and monkeys, obtained with stovaine (amylocaine hydrochloride, B. P.), in a maximal dose of 140 mg., lesions in the cord similar to those the authors obtained with procaine, except that in certain instances with minimal doses he obtained maximal effects. He stated that these degenerative changes in the

cord are due to the contact effect of the drug on the myelin sheaths. This explanation is not entirely satisfactory in the case of the authors' experiments, but they have nothing better to offer.

EDITOR'S ABSTRACT.

POSTVACCINAL ENCEPHALITIS. E. GORTER, J. A. M. A. **101**:1871 (Dec. 9) 1933.

Gorter reports a typical case of postvaccinal encephalitis, although cure was more rapid than usual. In not less than one third of all typical cases of the disease a fatal issue is observed. One of the most constant facts with regard to this type of encephalitis is the incubation period. In 108 cases recorded before 1929 this period was strikingly constant, the onset usually being observed on the tenth, eleventh or twelfth day after vaccination. It was often related in the history that the child was ill on the seventh or eighth day with fever and agitation but recovered more or less completely, so that the encephalitis was separated from the first period of disease by several days of relative well being. This course of events is, however, not seen regularly, because either the initial fever disappears on the seventh or eighth day or it progresses without interruption into the fever due to encephalitis. Among the symptoms headache was rather frequent and was often associated with vomiting. Drowsiness and even coma were often observed, whereas convulsions did not occur as frequently. Often meningitis was indicated by rigidity of the neck and a Kernig sign, and examination of the spinal fluid frequently confirmed the diagnosis of meningeal invasion by a high cell count, elevated pressure or a high sugar content. Paralysis of the ocular muscles or of the facial nerve or disorders of speech and deglutition were occasionally observed, whereas in other cases paralysis of the muscles of the legs and arms was present. Also in cases without paralysis the Babinski and similar reflexes were often seen, and the tendon reflexes frequently showed some abnormality. Death usually occurs within a week after the onset of the disease. In striking contrast with the bad prognosis as to life is the good prognosis with regard to sequelae. It is most exceptional for symptoms of a cerebral lesion to be seen after the disappearance of acute symptoms, and there is some reason to doubt the diagnosis of postvaccinal encephalitis in the rare cases in which a defective intelligence or hemiplegia remains after a disease resembling postvaccinal encephalitis. These characteristics are completely different from those of other forms of encephalitis or meningitis, whereas they are typical of a disease like tetanus.

EDITOR'S ABSTRACT.

SYPHILIS OF THE DIENCEPHALON AND ITS VEGETATIVE SYNDROMES. J. LHERMITTE, Ann. de méd. **33**:273, 1933.

Next to gliomas, syphilis of the diencephalon plays an important rôle in the production of manifold vegetative disturbances. If syphilitic inflammation of the adult brain is localized at the base, the tubero-infundibular syndrome is observed clinically: disturbances of water and fat metabolism, of sleep and of sexual function, each of which may predominate in the form of polyuria, polydipsia, adipositas, hypersomnia or amenorrhea. The sequence of appearance of the different signs may vary and may be combined with changing mental moods: capricious humor and melancholy.

The inclusion of the optic nerves in the disease process is revealed by amblyopia or amaurosis, by congestion or edema of the disks and by bitemporal hemianopia. Diplopia, transitory or lasting, and extra-ocular palsies indicate disease of the oculomotor nerves. The proof for the absence of a tumor is not difficult to establish. Headaches in syphilis are mild and rare. Marked papillary edema does not occur; instead, Parinaud's syndrome, Argyll Robertson pupils, ocular palsies and hemianopias are more frequent.

Among isolated syndromes of the tubero-infundibular region are mentioned: polyuria with elimination of amounts of urine as high as 20 liters during twenty-four hours; the syndrome of Mendel is a combination of polyuria with bilateral abducens paralysis; other combinations are polyuria with glycosuria (from 500 to 600 Gm.); retention of chlorides with edema; polyuria combined with adipositas. In rare cases hypersomnia may be the only early symptom, but in most cases it is combined with other vegetative disorders, as e. g., obesity, sexual impotence and amenorrhea. Hereditary syphilis affecting the diencephalon may produce syndromes similar to those described in the adult. Sometimes a basilar meningitis affecting the tuber cinereum may simulate a suprasellar tumor.

Specific therapy does not bring about a disappearance of all the vegetative disorders at the same time. In some cases polyuria disappeared first; in others it persisted and hypersomnia or amenorrhea disappeared following treatment.

WEIL, Chicago.

LOCALIZATION OF HEMICHOREA. YANNI TSIMINAKIS, Arb. a. d. Neurol. Inst. a. d. Wien. Univ. **35**:57, 1933.

Tsiminakis reports a case of hemichorea, of long duration, limited to the left upper and lower extremities. Necropsy revealed an old area of softening with extensive tissue destruction and cyst formation in the left cerebellar lobe. The vermis was unaffected, except for a small portion immediately adjacent to the affected cerebellar lobe. Except for some slight cellular changes, the ventral portions of both cerebellar lobes and part of the ventromesial portion of the dentate nucleus, corresponding to about one eighth of its size, were intact. The entire pathologic process was due to marked endarteritis and vascular sclerosis. As was to be expected, the contralateral olive was also involved; the pons showed small areas of softening, especially in the region of the right pyramidal tract; the left pyramidal tract showed no abnormalities. Owing to the complete degeneration of the left brachium conjunctivum, the red nucleus on the right side showed extensive demyelinization and marked reduction in size. Lesions were also observed in the frontal lobe, thalamus and basal ganglia on the right side, but they were not so marked as those in the cerebellum.

It is obvious that the pathologic process affected three systems, to any one of which the chorea could be attributed. The changes in the pyramidal tracts were too insignificant to play any rôle in the production of chorea; the same is true of the lesions in the right frontal lobe. The lesions in the basal ganglia, though more severe than those in the pyramidal tract and frontal lobe, were of recent origin and could not be held accountable for hemichorea of such long duration. On the other hand, the extent, intensity, age and characteristic localization of the pathologic process in the dentate nucleus and the almost total destruction of the brachium conjunctivum and the corresponding red nucleus point to the involvement of the cerebellar system as the most probable cause of the hemichorea. The case, therefore, is one of so-called Bonhoeffer's chorea, or chorea of the brachium conjunctivum.

KESCHNER, New York.

HEREDITY IN SYRINGOMYELIA. B. N. MANKOWSKY and L. I. CZERNI, Ztschr. f. d. ges. Neurol. u. Psychiat. **143**:701 (Jan.) 1933.

According to the theory of Bielschowsky and Henneberg, syringomyelia is a spongioblastosis which is dependent on the failure of the medullary canal to close properly. Henneberg also distinguished two types of failure to close: arraphia, due to the absence of the normal raphes, and dysraphia, due to inhibition in the approximation of the raphes. Spina bifida, diplomyelia and rachischisis belong to the first group; syringomyelia, hydromyelia, myelocystocele, spinal gliosis and glioma formation belong to the second group.

Reports of familial syringomyelia are few. Probaschenski (1899) reported cases in a father and two daughters; Nalbandow (1899) observed cases in a

mother and son. Clarke and Growes (1909) reported two cases in one family. Other cases have been reported in a father and son, in brothers and in a father and two sons. Bremer (1926) studied all the members, both healthy and diseased, in ten families with syringomyelia. In addition to one family with two brothers suffering from the disease, he found a number of anomalies among the "healthy" members, such as chicken breast, kyphoscoliosis, cyanosis and coldness of the distal parts of the extremities, enuresis, anomalies of the growth of hair, poor teeth and syndactylism. Shortly after this, Kino described cases of syringomyelia in a father, aged 66, and his three daughters, the localization of the process being the same in all the cases. Kino found also that five generations of this family had suffered from angiospastic phenomena in the fingers.

Mankowsky and Czerni report the cases of two brothers and of two sisters with syringomyelia, the two groups being in two different families. They believe that heredity is more common than is reported in this disease, and that these cases indicate that syringomyelia is a true heredodegeneration.

ALPERS, Philadelphia.

CROUZON'S DISEASE. J. NORDMANN and E. DUHAMEL, Rev. d'oto-neuro-opht. **11**:422 (June) 1933.

In 1912, Crouzon described hereditary craniofacial dysostosis as a distinct clinical entity, characterized by cranial deformity, facial malformations and ocular defects. Heredity plays a part, but not always, and it does not follow the mendelian law. The case is presented of a girl, aged 7, who had developed slowly. No similar deformity was discovered in the antecedents or among the brothers and sisters. Examination revealed a large protuberance at the superior edge of the frontal bone. A roentgenogram of the skull showed a cerebriform appearance of the vault, caused by irregular thinning of the osseous wall, giving a picture of the convolutions; the anterior fossa lay vertically; the sella turcica was deep and lay in a depression between the anterior fossa and the petrous bones; a curious fact was the opacity of the ocular globes. The facial expression was typical, but there was no prognathism. Exophthalmos of 23 mm. and left divergent strabismus with an angle of 15 or 20 degrees were observed, accompanied by bilateral white atrophy of the optic nerve. No other malformations were present; lumbar puncture and neurologic examination revealed negative results, and the blood calcium was normal.

Differential diagnosis between Crouzon's disease and oxycephaly is important. In the latter, the whole skull is distended upward, the anterior fossa is not so vertically placed, and the difference in level between the anterior and middle fossa is not so pronounced. In Crouzon's disease the enlargement is in the anterior part of the vertex. The points in common are exophthalmos, divergent squint and optic atrophy. Nordmann and Duhamel believe that intracranial hypertension causes the optic atrophy in oxycephaly by way of papillary stasis. Such an ophthalmic picture was described by Chatelin in a case of Crouzon's disease.

DENNIS, Colorado Springs, Colo.

EXPERIMENTAL COMPRESSION OF THE CENTRAL NERVOUS SYSTEM: CEREBRAL COMPRESSION PRODUCED BY THE INJECTION OF PARAFFIN AND BY PRESSURE WITH A DISK: HISTOPATHOLOGIC REPORT. CURZIO UGURGIERI, Riv. di neurol. **6**:593 (Dec.) 1933.

The author produced compression of the brain in rabbits by the subarachnoid injection of liquid petrolatum and by pressure with an extradural disk. He used ten rabbits and nineteen different stains in order to study all the histologic elements. Three rabbits with compression produced by the injection of paraffin were studied two, fifteen and sixty days after the operation; a rather marked, slow type of degenerative change was seen in the nerve cells, with proliferation of the astrocytic glia, while the microglia remained essentially unaffected. The

compression was rather slight. On the other hand, in the rabbits with more marked compression produced by means of a disk, of a duration of from two to one hundred and fifty days, the most pronounced changes were found in the nerve cells, atrophy and destruction ultimately resulting. The astrocytic glia showed an active reaction, with hyperplasia and hypertrophy. The microglia, in addition to hyperplasia, showed hypertrophy and transformation into numerous compound granular corpuscles. The nerve fibers showed degeneration and fragmentation. There was proliferation of the connective tissue in the meninges and in the vessels, and the formation of the compound granular cells with the phagocytic function tended to act as a substitute for the destroyed nerve tissue and to produce a cicatricial zone.

All these changes appeared to be directly proportional to the duration of the compression and were limited to the compressed zone and to its immediate vicinity; they resembled the pathologic changes found in human cases of cerebral compression by tumors, by external hydrocephalus or by subdural or extradural hematomas.

BARRERA, New York.

HYPOCALCEMIA: ITS RELATIONSHIP TO MIGRAINE. G. F. NORMAN, J. A. M. A. **102**:529 (Feb. 17) 1934.

Norman states that a few of the cases usually classified as migraine with respect to blood calcium determinations exhibit degrees of tetany varying from mild, almost imperceptible clinically, to severe. Hyperpnea in a few mild cases induced tingling of the extremities and occasionally a positive facial phenomenon. These conditions were quite constantly associated with low amounts of calcium in the blood. It is not assumed, however, that all cases of migraine exhibit these phenomena. As a matter of fact, cases of frank operative tetany that have been observed do not exhibit the characteristic symptomatology of migraine. Numerous cases are apparently associated with disease of the biliary tract. However, the failures of surgical measures on the biliary system to relieve the unilateral headache are only too well known. The question is raised as to whether the biliary disturbance depresses the calcium-regulating mechanism or, on the other hand, the calcium disturbance alone favors pathologic changes in the biliary system. For this complex the author proposes the term "migraine-tetanic syndrome," conforming to the idea that a degree of hypoparathyroidism exists and that the latter condition exists more frequently than is generally accepted. He assumes that symptoms of migraine and tetany are maintained dormant in this group by the calcium-regulating mechanism. One or the other complex may predominate. Whether unilateral headache and nausea with or without vomiting should be included in the picture recognized as tetany remains for further study. Norman observed about seventy patients complaining of periodic headaches associated with nausea and vomiting, and most of them have been improved by the oral administration of viosterol or the parenteral administration of potent parathyroid extracts.

EDITOR'S ABSTRACT.

ROUTES OF INFECTION IN OTOGENOUS MENINGITIS: A FULMINANT CASE OF HEMATOGENOUS ORIGIN THROUGH ANOMALOUS VASCULAR ANASTOMOSES. WILLIAM LAWRENCE GATEWOOD and NATHAN SETTEL, Arch. Otolaryng. **18**:614 (Nov.) 1933.

Meningitis may be caused by direct extension from the ear or by contact infection by metastasis. The more virulent the organism the less time the body has to acquire resistance. Infection attacks in order of frequency the mastoid process, the sigmoid fossa, the tegmen tympani and the tegmen antri, the pars petrosa, including the labyrinth, and the posterior wall of the semicircular canal and the posterior wall of the carotid canal. Infection may travel by way of the following routes: persisting sutures, vascular anastomoses, dehiscences, the labyrinth, the hiatus subarcuatus, the facial canal and the tip of the pyramid. The relative

frequency of these routes still remains, however, largely a matter of conjecture. Extension of infection by vascular or lymphatic routes is possible.

A case is reported, and the authors surmise that in it the infection may have been through the diploic vessels or through the lymphatics, and it may not have been by direct infection from necrosis of the dural plate which was suspected as a result of the roentgen examination. Autopsy was not done; therefore, the exact mode of extension could not be determined, but the interesting feature of the case was that the fulminating meningitis appeared following infection of the ear for two weeks. Signs of meningitis appeared suddenly after the otitis had subsided, and the patient died within twenty-four hours. Streptococci were found in the spinal fluid. The authors discuss the opinions of various writers as to the way in which infection reaches the brain from the ear, and come to the conclusion that in their case this was probably effected through the blood stream and by abnormal anastomosis.

HUNTER, Philadelphia.

THE PRESENT AND FUTURE OUTLOOK IN THE TREATMENT OF AMENTIA. L. PIERCE CLARK, Psychiatric Quart. **7**:50 (Jan.) 1933.

While not denying the physical basis of feeblemindedness, Clark believes that functional psychologic distortions, particularly defective libidinal distribution, are important in the development, understanding and treatment of intellectual defect. The normal person forms an identification with the person from whom he acquires knowledge: mother, teacher, nurse or friend. He "takes in" information by "ingesting" the teacher—as in the nursing period. At first there is no other way to acquire knowledge than to hold the thing being studied, to hold it in the mouth or hand. In the feebleminded, primary narcissism persists and forms a barrier against the forming of identifications.

Mentally defective persons should have the opportunity of living through the incomplete oral stages of development. The activities expressive of this stage (such as thumb-sucking and placing objects in the mouth) should not be too hastily restrained. To do so is to drive these impulses more deeply into the mind, where they may block later development. The teacher must establish a libidinal relationship with the pupil; when this is done the child will absorb what the teacher gives him as a manifestation of love. A further step is necessary: the libido projection must be displaced from the teacher to the work.

One of the difficulties with training feebleminded children is the danger of personality defect in the teacher. The rôle of the instructor is a superparental one, and the teacher's love life may become merged with the work to such an extent as to stimulate dependent clinging on the part of the pupil. For this reason only persons with normal libidinal life should be permitted to train mentally defective persons.

DAVIDSON, Newark, N. J.

NUMBER OF NERVE FIBERS IN THE ROOTS OF THE SPINAL CORD OF MEN. NILS ARNELL, Upsala läkaref. förh. **39**:97 (Dec.) 1933.

The work here reported was done on the initiative and under the supervision of Agduhr, who in 1919 showed that the ratio between the number of nerve fibers in the dorsal roots and the number in the ventral roots increases in a measure as the animal rises in the scale of the animal kingdom. According to Agduhr, the ratio is lowest in amphibians and highest in dogs. In mammals the ratio is greatest in the plexuses of the extremities. The material studied consisted of two spinal cords with preserved roots and nerve trunks of men, aged 30 and 55, respectively. The silver impregnation method of Agduhr (1930) was used. Arnell substantiates Ranson's observations concerning the presence of nerve fibers without myelin sheaths in all the roots of the spinal cord. They are particularly numerous in the dorsal roots. In the cord of the man aged 30, the nerve fibers in the dorsal roots on the left side numbered 952,777 and in the ventral roots on the same side, 197,256. The total number of nerve fibers in the dorsal roots on the right

side was 1,142,456, while that in the ventral roots was 228,862, with a ratio between the sensory and the motor fibers of 5:1. In the cord of the man aged 55, the total number of nerve fibers in the dorsal roots on the left side was 594,250, and in the ventral roots, 127,050, with a ratio of sensory to motor fibers of 4.7:1. The number of nerve fibers in both the ventral and the dorsal roots on the right side in a right-handed person is greater than the number on the left. Individual variations were not detected in the spinal cords of the two men. The ratio between the number of sensory and the number of motor fibers in men is much greater than that found by Agduhr in animals.

NOTKIN, Poughkeepsie, N. Y.

STUDIES IN EPILEPSY. F. L. MCLAUGHLIN, J. Ment. Sc. **79**:688, 1933.

In sixteen cases of epilepsy, the alkali reserve in the interval between attacks fell to within normal limits. The average for persons with epilepsy is 68.7 per cent by volume, and that for normal persons is 64.4 per cent. There is a fall in the alkali reserve during a fit; it is possible to correlate the clinical signs with this fall, which shows that the degree of the fall is related to the severity of the convulsions. The epileptic plasma tends to be slightly more alkaline than normal, but this can be seen only in a study of a large number of cases. The various changes in the blood are probably the result and not the cause of epileptic seizures. In the interval between fits, variations in alkali reserve and in lactic acid are usually within normal limits, but those of p_H extend to a range slightly above normal. The average concentration of lactic acid is normal. The average alkali reserve and p_H values are slightly above normal. The average of results obtained for alkali reserve and p_H during the two hour period preceding a fit show no significant difference from those of the interval period.

Results obtained from samples of blood of patients before, during and after a particular seizure lead to the following conclusions: (*a*) There is a fall in alkali reserve during and for varying periods after the fit, this fall being accounted for by the accumulation of lactic acid in the blood. A decrease in lactic acid during recovery is accompanied by an increase in alkali reserve. (*b*) The blood p_H decreases during the fit, owing first to apnea during the tonic stage and second to production of lactic acid during the clonic stage. Return of the p_H to normal is more rapid than in the case of normal persons after exercise.

This paper received the prize from the Royal Medico-Psychological Association in 1933.

KASANIN, Howard, R. I.

THE SIGNIFICANCE AND PATHOGENESIS OF THE CLIMACTERIC CRISES. G. MARANON, Rev. franç. d'endocrinol. **2**:313 (Oct.) 1933.

Maranon believes that the climacteric crises ("hot flashes") are pathologic manifestations of the climacteric. He claims that in "strictly physiologic women," except for suppression of menstruation, the climacteric passes without untoward symptoms. He adds, however, that a woman seldom reaches the critical age in a normal state. According to Maranon, the crises have a multiple pathogenesis. On one side, there are the neurohormonal factors consisting of an instability of the vasomotor centers, a condition which is facilitated by ovarian insufficiency and often by hyperthyroidism. In addition, the hypersuprarenalism with sudden discharges may release the attacks. However, there must be an additional factor to cause this complex neurohormonal state to express itself in the form of the well known crises. The additional factor is a pathologic condition of the cardiovascular system. Maranon advises looking for more or less pronounced lesions of the circulatory apparatus in women who suffer from severe crises, especially if the condition lasts longer than a year, and advises treatment for the cardiovascular system in women with climacteric signs. He built up his theory on the basis of Zondek's studies concerning the relationship between vasomotor

instability and dysfunction of the various endocrine glands, especially the ovaries, the thyroid and the suprarenal and pituitary glands. Maranon, however, believes that ovarian insufficiency alone is not sufficient to produce vasomotor instability; infantile women with hypoplastic ovaries, for instance, do not have climacteric crises. Maranon has no laboratory proof to offer for his theories. His reasoning is along logical lines, although it is not always convincing.

NOTKIN, Poughkeepsie N. Y.

ACQUIRED HYDROCEPHALUS: REPORT OF TWO CASES OCCURRING IN ADULTS. LYDIA PIERCE, Am. J. Psychiat. **12**:769 (Jan.) 1933.

Acquired hydrocephalus in adults is not common; it may be due to a constriction of the veins of Galen or, more frequently, to some impediment to the normal circulation of the cerebrospinal fluid. The underlying lesion may be neoplastic, congenital or inflammatory. Two cases are reported by Pierce; one was due to primary ependymitis, and the other, to some undetermined form of adhesive arachnitis or leptomeningitis. The first patient was a woman, aged 43, who had been an inmate of a state hospital for nine years. She was suffering from paranoid dementia praecox and had begun to deteriorate. However, there was no evidence of physical disease until a month before death, when she complained of backache. The temperature, pulse and respiration were normal. Death was sudden, and autopsy showed distention of the lateral and third ventricles, destruction of the septum pellucidum, enlargement and cystification of the choroid plexus of each lateral ventricle and pressure atrophy of the convolutions. The second case was of the communicating type; the patient, also a woman suffering from paranoid dementia praecox, was 49 years of age; she was mentally deteriorated but was in good physical health until thirty-six hours before death, when she had convulsions and fever. She died in coma; autopsy revealed distention of the lateral and third ventricles, with projection of the floor of the latter into the interpeduncular space, with consequent pressure on the optic chiasm. This was distorted and degenerated. The pia was thickened over the base and adherent to the cisterna magna. There was some injection of the subependymal vessels of the lateral ventricles, probably due to stretching.

DAVIDSON, Newark, N. J.

RETINITIS OF HYPERTENSION AND SPINAL DECOMPRESSION. A. MAGITOT and A. DUBOIS, Ann. d'ocul. **170**:534 (June) 1933.

Spinal decompression is valuable in treatment for exudative retinitis associated with hypertension. The loss of normal relation between the retinal and brachial tensions frequently found in this type of retinitis may be attributed to intracranial hypertension. The latter must be verified by Claude's manometer, by lumbar puncture or by suboccipital puncture. Decompression causes a rapid and permanent decrease of retinal arterial blood pressure. (If there is no decrease in this pressure, the retinitis will not improve.) Decompression is soon followed by the disappearance of violent and persistent headaches and by an improved general condition. The disk returns to normal, the hemorrhages are absorbed and the exudates are fragmented and diminish in number, in some cases disappearing completely. This improvement is accompanied by a return of visual acuity, which may be complete.

Three cases are presented. In the first, the brachial blood pressure was 260 systolic and 140 diastolic; urea, 0.42. The cerebrospinal fluid pressure was 41 by lumbar puncture when the patient was recumbent. The retinal arterial blood pressure was 140. Decompression was accomplished by lumbar puncture and then by fifteen injections of hypertonic dextrose serum, 30 per cent. Visual acuity improved from 1/50 and 3/50 to 7/10 and 5/10. The retinal arterial blood pressure became 100. With the hypertonic injections the general hypertension increased to 280 systolic and 130 diastolic. The other two cases were similar. Terson inquired whether lumbar puncture would not also be useful in certain cases of glaucomatous hypertension.

BERENS, New York.

BEHAVIOR OF UREA, URIC ACID AND CHOLESTEROL OF THE BLOOD IN THE COURSE OF MENINGO-ENCEPHALOPATHIES. N. LOLLI, Riv. di pat. nerv. **41**:96 (Jan.-Feb.) 1933.

Lolli reports on the behavior of urea, uric acid and cholesterol in the circulating blood in twenty-three cases of involvement of the central nervous system (hemorrhage in the brain, tumor of the brain, influenza, meningo-encephalitis, purulent meningitis, influenza meningitis, cerebral embolism and other conditions). The urea, uric acid and cholesterol in the blood and the urea and uric acid in the urine are often increased. The modifications in the proportion of urea have proved to be independent of involvement of the kidney or of a febrile reaction. There was an oscillation in the quantity of these components proportionate to the clinical oscillation of the disease; with recovery from the cerebral process there was a definite lowering of the figures.

Lolli thinks that there must be cerebral centers which regulate most of the chemical components of the blood. Therefore, he has studied experimentally the influence of lesions of the cortex and of the fourth ventricle in relation to variations of these components; he concludes that particularly punctures of the fourth ventricle produce definite variations in urea and cholesterol. At times these variations are even independent of the puncture itself and appear as a result of decompression following removal of fluid from the fourth ventricle. Experimental lesions of the cortex have caused slight but not constant variations. Probably the lesions act indirectly on the various regulating centers of the rhombencephalon.

FERRARO, New York.

SOME REFLEX CHANGES IN POLIOMYELITIS. JOHN A. TOOMEY, Am. J. Dis. Child. **46**:730 (Oct.) 1933.

The author studied three types of reflexes in a large series of cases, particularly in cases of poliomyelitis. The abdominal reflex was acutely active, except when the patient voluntarily held the abdomen tense, when he laughed or cried, when the abdominal layers were lax, when there was a massive fat pad over the abdomen and in new-born infants. He found that the reflex was modified or absent when the course of the spinal pathway was interrupted, as in multiple sclerosis, when tumors had stretched the abdominal wall, when there were infiltrations or inflammations in the abdominal wall, in certain cases of typhoid fever and intestinal disease and in some cases of meningitis. He found the reflex to be hyperactive in many cases of appendicitis, gastric ulcer, early acute peritonitis, disease of the gallbladder, renal colic, chorea, tabes dorsalis and parkinsonism. Very early in infantile paralysis he found some hyperactivity of the abdominal reflexes, which then became diminished or lost; later in the disease they were fairly consistently lost. He studied the cremasteric reflex, and found it to be present and essentially normal in poliomyelitis, except in cases in which there was evidence of a lesion at the reflex level. He, therefore, considered that the cremasteric reflex is of little value in the diagnosis of poliomyelitis but that the abdominal reflex is of considerably more value. He also studied the knee jerks and found that in some cases of poliomyelitis the knee jerks may be overactive, but that later they become diminished or lost. The sequence was so rapid in some cases that he thought it might easily have been unobserved.

WAGGONER, Ann Arbor, Mich.

A METHOD TO INTRODUCE LARGER AMOUNTS OF THE ARSPHENAMINES INTO THE CENTRAL NERVOUS SYSTEM. JOSEPH WITTENBERG, Am. J. Syph. **17**:339 (July) 1933.

To introduce larger amounts of arsenic preparations into the central nervous system, Wittenberg suggests the use of ephedrine, introduced intravenously before the injection of the arsenic. Ephedrine, by constricting peripheral vessels and raising blood pressure, increases the amount of blood in the brain, and deposits in that organ larger amounts of any drug which may be in the blood stream.

Two patients with neurosyphilis were studied to determine the correctness of this hypothesis. A solution of ephedrine, 1 grain (0.065 Gm.) to 1 cc. of distilled water, was injected intravenously. From thirty to fifty seconds was taken for the injection. Two minutes later, neoarsphenamine was introduced intravenously; this was given very slowly. One and a half hours later, lumbar puncture was performed and the spinal fluid was examined for arsenic content. Ten days after this procedure, a dose of neoarsphenamine was given without the preliminary injection of ephedrine. Examination of the spinal fluid was made under conditions identical with those followed at the previous treatment. The arsenic was measured by the Osterberg method. In the fluid removed after the injection of both ephedrine and neoarsphenamine traces of arsenic were found; in the fluid taken after the injection of neoarsphenamine alone no trace of arsenic was found. It would appear that the consecutive introduction of ephedrine and neoarsphenamine into the veins is safe, is likely to increase the effectiveness of arsenic therapy and is worthy of further study.

DAVIDSON, Newark, N. J.

THE PERSONAL TEMPO AND ITS INHERITANCE. IDA FRISCHEISEN-KOHLER, Character & Personality **1**:301, 1933.

Every person has his characteristic tempo which is expressed in all his doings, in acts of perception and volitional processes. Personal tempo is determined by tapping and metronome experiments—the person choosing the tempo most agreeable to him. Marked intra-individual constancy of personal tempo is found, environmental differences being associated with little alteration. On the other hand, a marked inter-individual difference of the personal tempo is demonstrated, but without reliable difference of tempo for different sexes and different ages and different social milieu. Variability of tempo of dizygotic twins corresponds almost exactly with that of brothers and sisters, and is considerably higher than variability of tempo of monozygotic twins. Percentage deviation of unrelated persons is considerably higher than of dizygotic twins and of brothers and sisters. This is taken as convincing proof of extreme dependence of the manifestation of the personal tempo on the inherited disposition. This is also indicated by the finding that the tempo of children is closely dependent on the tempo of parents. Two hypotheses as regards the mode of inheritance are discussed: inheritance depending on two allelomorphic series with dominance of the quicker tempo over the slower, or on one allelomorphic series with intermediate inheritance.

The author concludes that these results show that by comprehensive and statistical investigation of twins and families the inheritability of psychic characters may be demonstrated, and hypotheses formed as to the mode of their inheritance.

MATHER, Howard, R. I.

EXAMINATION OF DIATHERMY MACHINES FOR LOCAL DIATHERMY TREATMENTS AND REQUIREMENTS FOR ACCEPTANCE OF THESE MACHINES BY COUNCIL ON PHYSICAL THERAPY OF THE AMERICAN MEDICAL ASSOCIATION. ALLAN HEMINGWAY, J. A. M. A. **101**:776 (Sept. 2) 1933.

In treatment by diathermy, the sole therapeutic benefit results from the production of an increased temperature of the tissues treated. This requires that the frequency of the diathermic current be sufficiently high to prevent electrical stimulation and that associated with the high frequency current there shall be no low frequency surges of current, due to defects of the machine, which will excite the neuromuscular system. The machine must be so assembled as to insure (1) convenience of operation, inspection and repair, (2) ability to withstand moderately rough usage, as movement around a hospital, and (3) the complete safety of the patient and the operator from burns and shock, the faulty technic of treatment being taken into consideration. The author discusses the different parts of the typical machine for diathermy: the low frequency transformer, the voltage regulator, the low frequency leads and connections, the switches, the spark gaps, the con-

densers, the inductance in the high frequency oscillatory circuit, the milliammeter, the leads to the patient and the general assembly of the apparatus. The frequency test, test of high frequency energy output, test of machines for low frequency current surges, test of machine for operation and the efficiency of the machine are discussed under tests of the machine.

EDITOR'S ABSTRACT.

SCHIZOPHRENIA IN CHILDREN. HOWARD POTTER, Am. J. Psychiat. **12**:1253 (May) 1933.

Children, lacking the adult faculty for putting their thoughts into proper words, express delusional trends in a naive manner. As might be expected, their psychotic manifestations are largely affective and behavior disorders. Potter reports six cases of dementia praecox in children under the age of 12, none of whom showed physical evidences of puberty. The youngest patient was a boy, aged 4, who displayed laughing spells, abstraction and mutism. From infancy he was active and bright but indifferent to other children. At the age of 3½, he began to be irritable, cross and destructive and was disinterested in the activities and questions of those about him. He spent most of his time in bed, whispering to himself and playing with his fingers. His talk was meaningless; his answers were irrelevant. His face was fixed and blank. During his stay in the hospital he had occasional fits of destructive rage; at times, however, he displayed true catatonic waxy flexibility. He failed to improve during this period, and a diagnosis of catatonic schizophrenia seemed justified. Nine other, somewhat similar cases are cited. In most of these the dementia praecox was of the catatonic type. The ages of the patients ranged from 4 to 12. In no case was any outstanding improvement noted during the course of the stay in the hospital. Potter suggests that dementia praecox is more common in childhood than is generally supposed; he believes that careful psychiatric study of many so-called feebleminded children will disclose cases of schizophrenia.

DAVIDSON, Newark, N. J.

THE KAHN REACTION IN THE AQUEOUS HUMOR. F. BRUCE FRALICK, Arch. Ophth. **10**:745 (Dec.) 1933.

Relatively recent developments in microchemical laboratory technic have stimulated renewed interest in the characteristics of the intra-ocular and other body fluids. First the blood and then the spinal fluid occupied medical investigators in determining the specificity and adaptability of this test for syphilis. Various investigators have worked with different fluids. Some used urine; others, the milk of syphilitic women, and others, the serum from artificially formed blisters, ascitic fluid and the fluid from the joints in syphilitic arthritis. Von Lussich-Malkovich was apparently the first to consider the advisability of performing the Wassermann test with the aqueous humor. Since then a fair amount of work has been done. On the basis of this work and of that done by the author himself, it is possible to substantiate the hypothesis stating that a positive Kahn reaction in the aqueous humor may be expected when the tissues from which that fluid is derived are syphilitic and that a negative reaction may be expected with a nonsyphilitic involvement of these tissues. No correlation was observed between the Kahn reactions of the spinal fluid and those of the aqueous humor. The most interesting exception to the findings was found, as could be expected, in inadequately treated syphilitic patients showing iridocyclitis, in whom the reaction of the aqueous was positive and that of the blood and spinal fluid was negative.

SPAETH, Philadelphia.

THE PRECIPITATION OF ENDOGENOUS PSYCHOSES BY INCIPIENT DEMENTIA PARALYTICA. O. OESCHEY, Arch. f. Psychiat. **100**:46 (June) 1933.

Following up work of Bostroem on this problem Oeschey reports an investigation of eleven cases of dementia paralytica treated by malaria in which the nature of the psychosis was closely related to the clinical pictures seen in manic-depressive

psychosis and in schizophrenia. Eight of the patients belonged to the manic-depressive group. In all the signs appeared early in the process, accompanied by very little dementia and by definite pictures of either depressions or manias. The treatment was successful and the process cleared up, leaving either no paretic defect or very little. The case histories showed a large proportion of persons whose previous personality had been of cyclothymic type, and it would appear that the organic process served only as a precipitating factor in the condition. The remaining three cases showed clinical pictures closely related to the schizophrenic type. Here the malarial treatment helped in clearing up the organic psychosis, but the patients either were left with a schizophrenic defect or went on to schizophrenic deterioration. In discussing these findings the author expresses the belief that the recognition of the fact that one is dealing with an endogenous psychosis in such cases helps in determining the prognosis and is also of great assistance in recognizing the condition at an early stage. Malarial treatment is especially successful in persons who suffer from the benign psychoses. MALAMUD, Iowa City.

THE HISTOPATHOLOGY OF THE RHOMBENCEPHALON IN ORGANIC DISEASES OF THE CENTRAL NERVOUS SYSTEM, WITH PARTICULAR REFERENCE TO THE LOCALIZATION OF THE VEGETATIVE NUCLEI. MARTHA DANCZ, Ztschr. f. d. ges. Neurol. u. Psychiat. **147**:555, 1933.

1. In various organic diseases of the central nervous system the rhombencephalon shows involvement which is more or less constant according to the type of disease.

2. The bulbar origin of hypertension of the central nervous system is not likely, since neither vessels nor cells show specific changes and the intensity of the changes does not parallel the degree of hypertension. There are ectodermic and mesodermic changes in cases without hypertension.

3. The bulbar anemia consequent on the arteriosclerotic changes cannot be considered the important factor in the origin of hypertension, since in cases without hypertension endarteritis obliterans may be present and may be much more marked than in arteriosclerosis.

4. On the basis of the focal intense cell loss in the vegetative vagus nucleus, with the clinical vasomotor manifestations, one must consider the dorsal nucleus of the tenth nerve to have a vasomotor rôle.

5. There is doubt about a respiratory center in the locus caeruleus, since severe changes have been found there without respiratory changes.

WINKELMAN, Philadelphia.

SOMNIFAINE NARCOSIS: TOXIC SYMPTOMS AND THEIR TREATMENT BY INSULIN. ROLF STRÖM-OLSEN, J. Ment. Sc. **79**:638, 1933.

The author follows the Kläsi technic with his own modifications in using a barbital derivative. The drug was injected intramuscularly, with an initial dose of 2 cc. Then from 5 to 6 cc. was administered in twenty-four hours, which was sufficient to produce a fairly deep narcosis. The present report covers a series of forty cases, in none of which were fatalities observed, although one patient contracted influenza at the termination of the course and died two weeks later of pneumonia. The most important toxic symptoms are ketosis, with diminished dextrose tolerance, decrease of blood pressure, tachycardia, circulatory collapse, oliguria, albuminuria, disturbances of coordination, including difficulty in swallowing, pyrexia and cutaneous eruptions of an erythematous type. Other changes to be noted are leukocytosis, a fall in the blood calcium and a slight increase in the blood urea. Epileptiform convulsions did not occur in the present series. The most serious problem is the disturbance of the carbohydrate metabolism, with the development of ketosis. This was controlled by the administration of insulin and dextrose during the narcosis. The author advises that insulin and dextrose be

administered in the beginning of the treatment, as complications do not develop in such cases, whereas in other cases in which this is not done complications are liable to occur. Myocarditis, arteriosclerosis, undernutrition and respiratory disorders are the chief contraindications to the use of the barbital derivative.

KASANIN, Howard, R. I.

HISTOPATHOLOGIC CHANGES IN THE NERVOUS SYSTEM IN THALLIUM POISONING. E. SCHARRER, Ztschr. f. d. ges. Neurol. u. Psychiat. **145**:454 (April) 1933.

Thallium, which has been used recently as a depilatory, may cause changes in the nervous system. Scharrer reports a case of thallium poisoning in man, together with experiments carried out on monkeys. The case was that of a man, aged 41, who had taken rat poison. In the course of from five to eight days, his hair fell out; later he had pains, paresthesias and feelings of coldness. Examination revealed polyneuritis. Later, there developed disturbances of speech and gait, and the patient died. Necropsy revealed degenerative changes in the spinal cord, characterized by loss of myelin sheaths and fatty degeneration. This was most marked in the cervical, thoracic and lumbar regions and least in the sacral. The degeneration of the posterior columns was most marked in the thoracic region. The cells of the dorsal vagus and vestibular nuclei showed axonal degeneration. The inferior olivary bodies showed a loss of cells, the remaining cells presenting a change similar to that of retrograde degeneration. Most of the cells of the median accessory olivary body were in this state. The dentate nucleus showed changes. In some parts fatty degeneration of the cells was marked; in other parts it was less pronounced. Glial hyperplasia was present. There was no evidence of a pathologic process in the cortex or white matter of the cerebrum, in the basal ganglia or in the cornu ammonis.

ALPERS, Philadelphia.

THE DERMATOMES IN MAN. O. FOERSTER, Brain **56**:1 (March) 1933.

In reviewing the methods for defining the dermatomes in man, Foerster states that the vasodilatation following electrical stimulation of the posterior roots, which carry efferent as well as sensory fibers, is the result of stimulation of these fibers. The observations presented in this article are the result of thirty years of experience in neurosurgery and offer confirmation of Sherrington's researches on the dermatomes in the monkey and of Head's scheme of the dermatomes in man. With the "isolation method" (division of a series of contiguous roots above and below a single root which is preserved), it was possible for the author to define all the dermatomes of the lower extremity and the sixth cervical dermatome. With the "constructive method," the other cervical and thoracic dermatomes were defined. Vasodilatation following stimulation of nearly all cervical, thoracic, lumbar and sacral posterior roots offered further corroboration. Ninety-three figures are used to illustrate this enormous amount of clinical material. Some of the conclusions are: 1. The tactile dermatomes are larger than the pain and the thermal dermatomes. 2. When all filaments except one of each of a number of roots are cut, no anesthesia results and each of the corresponding dermatomes preserves its sensibility. 3. The difference between the results of experimental physiologic studies in animals and clinical observations in man lies in the far greater difficulties presented by human material.

MICHAELS, Boston.

THE INFLUENCE OF CERTAIN STATES OF CONSCIOUSNESS ON RESPIRATORY ARRHYTHMIA. W. FRANKE-STEHMANN, Arch. f. Psychiat. **98**:787 (Feb.) 1933.

By respiratory arrhythmia is meant an irregularity of the pulse in which each pulse beat is normal but the pulse frequency is increased with inspiration and decreased with expiration. In normal persons there is a definite relationship between the respiration phases and the pulse frequency. It may change, however,

in certain pathologic conditions. The physiologic mechanisms of this arrhythmia are not as yet definitely agreed on; some authors are of the opinion that they are to be looked for in the special relationship between the respiratory and the circulatory centers, whereas others think that the arrhythmia is a product of mechanical disturbances within the thorax. A number of observations have been made which show that certain changes in mental functions may cause deviations from the normal or physiologic arrhythmia.

Franke-Stehmann studied patients suffering from different types of mental disturbances. He reports the following observations: patients with blunted and shallow emotional tone show a more pronounced respiratory arrhythmia than do restless, irritable and apprehensive persons. He agrees with Wiersma that a chronically increased state of irritability or mental activity tends to decrease the arrhythmia. He has found no special relationship between certain disease entities and changes in arrhythmia, so that diagnostically the symptom is of no value.

MALAMUD, Iowa City.

THE POSTTRAUMATIC NEUROSES. G. W. HALL and R. P. MACKAY, J. A. M. A. **102**:510 (Feb. 17) 1934.

Hall and Mackay point out that a study of a great number of cases of posttraumatic neurosis reveals at once that the posttraumatic neuroses do not differ in any essential way from most other, nontraumatic neuroses. They can all be divided into groups of considerable clinical value, according to their outstanding symptomatology. The majority of cases may be classified into posttraumatic neurasthenia, posttraumatic anxiety neurosis and posttraumatic hysteria. The authors give the principal clinical features of these three types of neuroses and discuss the psychology of the posttraumatic neuroses. Under treatment, they state that the patient should not be antagonized and that financial matters (of treatment) should be settled at once and not in protracted payments by which the question of the patient's disability is constantly kept open. The patient should be put to some form of work so that his interest is centered on something besides himself. Medication and various forms of physical therapy or electrotherapy are useful in giving him reason to expect improvement and a basis for relinquishing his symptoms. That such treatment may lack a physiologic rationale, in view of the fact that no physical basis exists for his symptoms, is neither here nor there. The psychologic rationale is ample warrant for such procedure. With these methods, encouragement and optimism find an important place when used tactfully and reasonably.

EDITOR'S ABSTRACT.

EXPERIMENTS ON THE FEEDING BEHAVIOR OF THE OYSTER, OSTREA GIGAS. A. E. HOPKINS, J. Exper. Zool. **64**:495 (Feb. 5) 1933.

A method is described whereby the relative rate at which the oyster pumps water can be recorded continuously on a kymograph along with movements of the shell. The amount of water pumped at constant temperature is determined by the degree of openness of the shell, which in turn depends on temperature. The shell is open widest at about 20 C. No water is pumped until the shell is open more than a distance equal to the thickness of the borders of the mantle at the edge of the shell. The optimum temperature for the activity of the cilia of the gills appears to be above 25 C. At about 20 C. and above, the shells begin to close and serve as a brake on the activity of the gills. Because of this behavior, the optimum temperature for the pumping of water or feeding, considering the animal as a whole, is about 20 C. The border of the mantle is divided into two lamellae, the inner of which is capable of moving in such a manner as to interfere with or stop the flow of water produced by the gills. The activity of the musculature of the gills, the flattening the gills together or against the adjacent mantle and the contraction of the gills as a whole give rise to large variations in the

rate of flow. The reaction system concerned in the pumping of water involves gills, adductor muscle and mantle, which appear to function with a large degree of independence of one another.

WYMAN, Boston.

A METHOD OF ESTIMATING THE FUNCTIONAL ACTIVITY OF THYROID BY MEANS OF URINE OR SERUM. MUNINYONG KOH, Keijo J. Med. **4**:1 (March 31) 1933.

A review of the literature of thyroid experimentation reveals the presence of the A neutralizing substance in the urine or serum of an animal, decreasing after thyroidectomy, but increasing as a result of feeding with thyroid extract. Fluctuation in the amount of the substance is parallel with that of the thyroid hormone. Its identity with the thyroid hormone is not yet fully established, for the A neutralizing substance decreases after thyroidectomy, and is later slightly restored by physiologic processes. In the urine of the normal rabbit the author finds a substance that neutralizes the mydriatic action of A. The rabbit fed with powdered thyroid has a markedly increased quantity of A neutralizing substance in both the serum and the urine. The substance does not exist in the thyroid powder, but appears only when the powder is introduced into the animal body. The substance is increased in the urine in pregnancy and in exophthalmic goiter. It is not a hormone itself, but changes in amount parallel with that of the thyroid hormone. It is easily soluble in water, is insoluble in alcohol or ether, and is stable to heat up to 80 C. (176 F.) for thirty minutes, but is destroyed at 100 C. (212 F.). It occurs in the serum combined with albumin, and cannot be separated by absorption with silicious earth.

HART, Greenwich, Conn.

THE OPTIC NERVE AND TRIVALENT AND PENTAVALENT ARSENIC PREPARATIONS. A. LACROIX, Ann. d'ocul. **170**:581 (July) 1933.

Lacroix states that the complications of arsenotherapy and particularly the lesions of the optic nerve depend on certain facts, knowledge of which often enables one to foresee accidents and avoid arsenic medication. These facts are: 1. Arsenic salts vary. Arsphenamine, with mercury, should be used in ordinary practice. In cases of intolerance or accident it should be replaced by bismuth. Pentavalent preparations of arsenic should not be used in the treatment of hereditary syphilis on account of their elective toxicity for the optic nerve and their lower effectiveness. They should be reserved (sodium acetarsone or tryparsamide) for certain cases of serious nerve syphilis or even for hopeless cases (dementia paralytica). Even in these cases, many authorities prefer malaria therapy. 2. The maximum dose varies according to the preparation; injections should be made at sufficiently frequent intervals to avoid neurorelapse. 3. There may be general and local contraindications (visceral lesions, lesions of the optic nerve in aged patients). 4. Observation of the patient during treatment and examinations of the eye are essential.

It cannot be stated definitely that there is no possibility of injury to the optic nerve if the facts cited are known, for the importance of personal factors often cannot be foreseen. The results and reactions which occur during the treatment must be observed constantly.

BERENS, New York.

FLACCID PARALYSIS IN CEREBRAL HEMIPLEGIA. W. A. STARKES, Ztschr. f. d. ges. Neurol. u. Psychiat. **144**:418 (Feb.) 1933.

Starkes analyzes the factors producing flaccidity in cases of hemiplegia in which the paralysis remains flaccid for long periods. Among the cases reported were three in which flaccidity persisted for one year, ten years and four months, respectively. Oppenheim observed in cases of large cortical and subcortical lesions persistent flaccid hemiplegia which developed into complete hemiplegia and hemianesthesia. Fischer supported the hypothesis that there is a close relationship between flaccid paralysis and anesthesia. Wallenberg attributed the flaccidity

to lesions in the tegmentum. Bing and others have expressed belief that it is due to a cortical lesion. Foerster stated that in flaccid paralysis there is a cessation of the stretch reflex. The spinal component of the stretch reflex is well known, since high cervical transverse lesions produce a flaccid tetraplegia. There is likewise a cerebellar component, the occurrence of long-standing hypotonia in cerebellar disease being well known. Furthermore, Foerster showed that acute conditions of the pallidostriate system lead to flaccid paralysis. In the cases of Starkes there was no involvement of cerebellar and spinal components. Furthermore, there was no anesthesia or other sensory disturbance. Because two of the four cases were accompanied by choreo-athetotic movements, it is assumed that the hypotonia is due to a disturbance in the function of the subcortical ganglia.

ALPERS, Philadelphia.

SYMPATHECTOMY FOR INTRACTABLE PAIN IN INOPERABLE CANCER OF CERVIX. J. P. GREENHILL and H. E. SCHMITZ, J. A. M. A. **101**:26 (July 1) 1933.

At present there are only two satisfactory ways of relieving the intractable pain that is associated with inoperable malignant tumors in the pelvis; both are surgical. One, chordotomy, requires an extensive laminectomy, and unless the incision in the cord is accurately placed the pain may not be completely relieved or paralysis may result. The second operation, sympathectomy, may readily be performed by any gynecologist. The risk involved is slight, the operation is technically not difficult, local infiltration may be used for at least part of the procedure, and the operation enables one to inspect and palpate the carcinomatous involvement. The operation that the authors advocate for the pain associated with inoperable carcinoma of the cervix consists of resection of that portion of the sympathetic system known as the superior hypogastric plexus or the presacral nerve and also the two hypogastric nerves. The results obtained in thirteen cases were remarkable, because all the patients experienced instant and complete relief from their suffering. The authors believe that this operation should be performed on almost all women who suffer constant and excruciating pain as the result of inoperable cancer of the cervix.

EDITOR'S ABSTRACT.

DIATHERMY IN THE TREATMENT OF GENERAL PARALYSIS AND IN WASSERMANN-FAST SYPHILIS. JAY FRANK SCHAMBERG and THOMAS BUTTERWORTH, Am. J. Syph. **16**:519 (Oct.) 1932.

This article is a report of treatment with diathermy in seven Wassermann-fast cases of visceral syphilis and nineteen cases of syphilis of the central nervous system in its various forms. The apparatus recommended by Neymann was used, except that a many-tailed binder replaced the cumbersome canvas jacket. Electrodes were applied to the back, chest and abdomen. Current intensities varied from 3 to 6 amperes. The current was discontinued when the temperature of the mouth reached 105 F. Treatments lasting for from four to seven hours were given on alternate days, from six to sixteen elevations of temperature being produced in each patient. Over a fourth of the patients suffered from burns under the electrodes. Two thirds of those who received this course of treatment improved, half of them enough to return to their former occupations. Three patients with tabes were in the series; one became worse, and one was relieved; the third had less pain, but was objectively unimproved. The seven patients with Wassermann-fast visceral syphilis showed no serologic or clinical improvement.

DAVIDSON, Newark, N. J.

THE TUBER CINEREUM AND ITS RÔLE IN THE PRINCIPAL FUNCTIONS OF METABOLISM. WATER, SUGAR AND FAT METABOLISM. G. ROUSSY and M. MOSINGER, Ann. de méd. **33**:193, 1933.

The intimate relationship between tuber cinereum and hypophysis is discussed, together with the regulation of the manifold vegetative functions which have been attributed to the latter. It now seems well established that water, fat

and sugar metabolism are under the influence of the ganglionic centers of the tuber cinereum. The former theories on the predominance of the floor of the fourth ventricle in the regulation of these metabolic processes have been abandoned in favor of the dominating influence of the tuber cinereum. In addition, the vasomotor functions of arteries and capillaries are regulated by its nuclei. The hypothalamus may be considered as the primary center for the regulation of metabolism, while the neothalamus and neocortex are associated centers. In psychologic terms, therefore, one may agree with Camus and Roussy and consider the tuber cinereum as the center for primordial instincts, thirst, hunger and sex, and the hypothalamus as the regulating center for the "instinct" ("l'être instinctif").

WEIL, Chicago.

CLASSIFICATION OF CASES OF RETROBULBAR NEURITIS. G. WEILL, Ann. d'ocul. **170**:696 (Aug.) 1933.

Having made the distinction between chronic retrobulbar neuritis of an alcoholic-nicotine origin and true optic neuritis, Weill considers cases of retrobulbar neuritis according to a definite classification, that is, without ophthalmologic examination. There are two classes. The first class consists of the typical cases, which are generally unilateral and have an acute course; they usually occur in persons between the ages of 15 and 35. This form of retrobulbar neuritis is almost always a symptom, and often the first symptom, of multiple sclerosis. The second class consists of atypical, subacute cases, sometimes bilateral; they present, besides central scotoma, peripheral and often temporal contraction of the visual field. Weill affirms that the acute, typical forms are cured spontaneously and without intervention. The atypical forms, on the contrary, may be favorably influenced by operation. In addition, Weill states that many cases of atypical retrobulbar neuritis are caused not by sinusitis but by diseases of the sella turcica and particularly by tumors which surround the optic nerve.

BERENS, New York.

MENINGITIS DUE TO THE INFLUENZA BACILLUS OF PFEIFFER (HAEMOPHILUS INFLUENZAE): A STUDY OF ONE HUNDRED AND ELEVEN CASES, WITH FOUR RECOVERIES. J. B. NEAL, H. W. JACKSON and EMANUEL APPELBAUM, J. A. M. A. **102**:513 (Feb. 17) 1934.

Neal, Jackson and Appelbaum correlate certain data in regard to 111 cases of meningitis due to the influenza bacillus under their observation. This disease is essentially one of young children. The evidence points to the fact that it is usually a primary disease and that it is caused by strains of influenza bacilli that tend to fall in one group. The pathologic examination usually shows severe toxic changes in the viscera. There is no particular tendency to the development of adhesions in the meninges or to the formation of abscesses in the brain. Of the 35 cases in which recoveries have been reported, 4 have been under the authors' observation. At present there is no adequate method of treatment. Further research should be done toward the development of a more potent serum with high antitoxic as well as antibacterial properties. Serum may be given early, intravenously as well as intraspinally. EDITOR'S ABSTRACT.

DIAGNOSTIC, PROGNOSTIC AND THERAPEUTIC NERVE BLOCKS. H. S. RUTH, J. A. M. A. **102**:419 (Feb. 10) 1934.

Ruth states that through many experimental and clinical studies, both in this country and abroad, there has evolved a technic for injections into the majority of the important components of the nervous system, without open section. With this accepted and definite technic available, regional anesthesia has become increasingly efficient and popular. Another result has been the application of these practices as a valuable adjunct to the practice of medicine and surgery along diagnostic, prognostic and therapeutic lines. At the present time, these methods

are used in numerous conditions, and the reported results are frequently contradictory and confusing. The author attempts, by a review of the literature on the subject, together with personal observations covering two years of use of such injections to estimate their present status. He describes the technic of injection, discusses the pathologic changes produced by the injection of alcohol and the diagnostic and prognostic block and considers the effect of injections to block the nerve in aortic aneurysm, bronchial asthma and vasospastic, malignant and miscellaneous conditions.

EDITOR'S ABSTRACT.

SEROUS MENINGITIS OF ALLERGIC NATURE. J. H. SHELDON, Lancet **1**:798 (April 15) 1933.

A case is reported in which an attack of acute tonsillitis was followed after an interval of eight days by a generalized scarlatiniform rash, and by two groups of symptoms which developed concurrently; (1) a series of involvements of the larger joints, characterized by pain and effusion, the latter being especially prominent in the knees, and (2) serous meningitis, of sufficient severity to give rise to 4 diopters of papilledema. Both groups of symptoms subsided after a peritoneal abscess caused by a hemolytic streptococcus developed; its drainage led to complete recovery. The diagnosis of hydrocephalus caused by serous meningitis was established by the normal character of the cerebrospinal fluid, its increased pressure and the subsidence of symptoms, including the papilledema, after two lumbar punctures. The nature of the effusion into the joints was not established by the puncture of a joint, but its complete subsidence in every case indicated that the fluid was certainly not purulent, and it was, therefore, assumed to have been serous.

BECK, Buffalo.

THE RELATIONSHIP BETWEEN THE LIPASE CONTENT AND THE NEUROTOXIC EFFECT OF SERUM AFTER EXPERIMENTAL INJURY OF THE LIVER. A. WEIL and L. CRANDALL, Ztschr. f. d. ges. Neurol. u. Psychiat. **140**:577 (July) 1932.

About four days after injury of the liver by tying the gall or pancreatic ducts, substances appear in the blood stream which can be demonstrated in a test tube and which exert a destructive effect on the spinal cord of rats. These toxins are not identical with the simultaneously increased lipases. They are heat-resistive, and can be demonstrated after several weeks when the lipases have returned to normal. These toxins get into the ventricles through the choroid plexus, or they disseminate into the cortex by way of the cerebral vessels. Their greatest effect is at the point of dissemination and of greatest concentration. Here they produce in some cases a spongy tissue necrosis in the ventricle wall, or demyelinization foci in the spinal cord. All cases showed a widespread disease of the ganglion cells. A dense glial proliferation was found in the demyelinated areas. The mesenchyma is not actively involved.

ALPERS, Philadelphia.

NORMAL RESPIRATION AND THE INFLUENCE OF THE VAGI. E. S. SCHAFER, J. Physiol. **75**:130 (June 21) 1932.

Respiration consists of inspiration and expiration; normally there is no pause. No definite relationship obtains between the frequency of the pulse and that of quiet respiration, but usually there are from five to eight heart beats to each respiration. Expiration is never a passive process, but is always accompanied by muscular activity. Although the regular rhythm of respiration is caused by inflation and deflation of the pulmonary alveoli, acting through the vagi, it is assisted by afferent impulses emanating from the respiratory muscles. If the vagi are severed, these impulses may be sufficient to maintain a normal rhythm. It is doubtful if the respiratory center has a rhythm of its own, independent of all reflexes. In the experiments which have been thought to prove its automatism, the afferent impulses from the respiratory muscles have not been taken into account.

ALPERS, Philadelphia.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY AND NEW YORK ACADEMY OF MEDICINE, SECTIONS OF NEUROLOGY AND PSYCHIATRY

Joint Meeting, Jan. 9, 1934

CLARENCE P. OBERNDORF, M.D., *President, in the Chair*

CLINICAL MANIFESTATIONS OF FEMALE SEXUAL DEVIATIONS. DR. ADOLPH STERN.

A review of the literature on female sexual development and its bearing on the clinical manifestations of neurosis in women reveals a gradual accumulation of facts and theories.

It has been accepted that the roots of neurotic illness in the male lie in the pathologic states of sexual life. The crucial period coincides with the attempts of the ego to resolve the Oedipus complex by the repression of the sexual impulses directed toward the mother. The ego is impelled to defensive behavior through fear of castration; the ego seeks to protect the genital pleasure-giving and narcisistic penis. Castration anxiety in the male is the impelling affect at the basis of the ego's efforts to resolve the Oedipus complex.

So clearcut a situation does not exist in connection with the origin of neurosis in the female. Confusion was caused by the theory that the mother is also the first libidinal object to the female. Until recently it was generally held that the modality of these libidinal strivings was masculine, by virtue of the little girl's attitude toward her clitoris, namely, that she behaved toward it as the boy did toward his penis. To become a female genitally the little girl first had to overcome her masculine attitude toward her mother. Another factor that offered difficulty in the understanding of the origin of neurosis in the female was the theory that in the female the castration complex initiated the Oedipus complex. This is the opposite to the condition which obtains in the male. A third factor was concerned with the rôle of penis envy in the female. That this was a reaction to the castration complex and that it came into being when the girl noticed the difference between her genitals and those of a boy were evident enough. But the clinical evidences of marked anxiety in connection with the various phenomena of the castration complex in the female were not sufficiently well explained on the basis of penis envy alone, so far as penis envy is a product of narcisistic yearning. This point is emphasized by Rado in an article in the *Psychoanalytic Quarterly* (**2**:425 [July-Aug.] 1933).

Changes in theory took place for some time. The older concepts, as briefly outlined, gradually underwent modification. Analysts questioned the long held belief that the little girl was unaware of the existence of the vagina; again they began to doubt the theory that the first phase of the female sexual life was masculine. As a corollary to this the question naturally arose as to whether the father was not the first love object of the girl, as the mother was of the boy.

This change in attitude brought the problem of the castration complex in the female into the foreground. Much has been written on this subject; a good deal of this material still stands on a firm basis. More emphasis on the rôle played by castration anxiety in the problem of neurosis in the female was needed. In the case of neurotic illness in the male this had long since assumed a central position.

Having the foregoing factors in mind, one is in a better position to understand Rado's contribution to the elucidation of neurotic illness in the female. His starting point is the problem of castration fear. This begins in the girl when she notes the difference between her genitals and those of a boy. At this time she is already in the positive phase of the Oedipus complex and is indulging in masturbation. The sight of the male genitals acts in a "traumatic" way; she ceases masturbation, feels herself injured and inferior, turns in on herself and indulges in painful fantasies of being injured, into which a pleasurable element is injected. The latter is due to the masochistic replacement of the previous genital pleasure. The girl now is in a masochistically distorted "feminine position." Because the ego senses danger from these new instinctive (masochistic) impulses, it undergoes repression. By way of compensation, an "illusory penis" is created. The masochistic impulses are dangerous to self-preservation and therefore "ego-alien." Defense against them on the part of the ego by means of the illusory penis is not effective, because the sense of reality renders the illusion negative. The illusory penis also undergoes repression, in order to be removed from the influence of reality. The various familiar phenomena of displaced castration fear result. From this point on, neurotic illness can be traced; castration fear becomes the motive power impelling the ego to protect itself against the instinctive danger of masochism.

It is impossible to do justice to Rado's contribution in an abstract. The article throws much light on the problem of neurosis in the female.

PSYCHIC SUICIDE. DR. A. A. BRILL.

Psychic suicide may be defined as suicide committed without the use of physical agents. It is questionable whether it is possible for one to die by the simple "will to die." This statement is based on recent investigations of suicide by Freud and his pupils, notably Menninger, who stated that some day it may be possible to show that conversion hysterias and some forms of organic disease represent chronic focal self-destructive attacks, while asceticism and martyrdom represent chronic generalized self-destruction.

There are cases in church history and in practice in which people have died apparently without the intervention of any physical agent. Thus, the priest Basil volunteered to be the first to celebrate the dedication of a sepulcher and died as if "falling asleep." Numerous cases have been reported also in the press and in literature in which persons died, plainly showing that they no longer had the "will to live." Many explorers have reported that primitive people can "will to die" and do so; in numerous cases autopsy has revealed no cause for such deaths.

Hysterical bleeding, as manifested in ulcers of the stomach and vicarious menstruation, may possibly be associated with stigmatizations. Such cases have been reported repeatedly by the Catholic Church; a number of cases have been studied by competent physicians and have been found to be authentic.

Recent studies of the relation of the soma to the psyche may explain such cases; in a number of cases from my own practice the patients seemed to have had the premonition that they would die, and actually did die; some unconsciously realized that their end was coming (unconscious insight), feverishly finished all their affairs, and died soon thereafter.

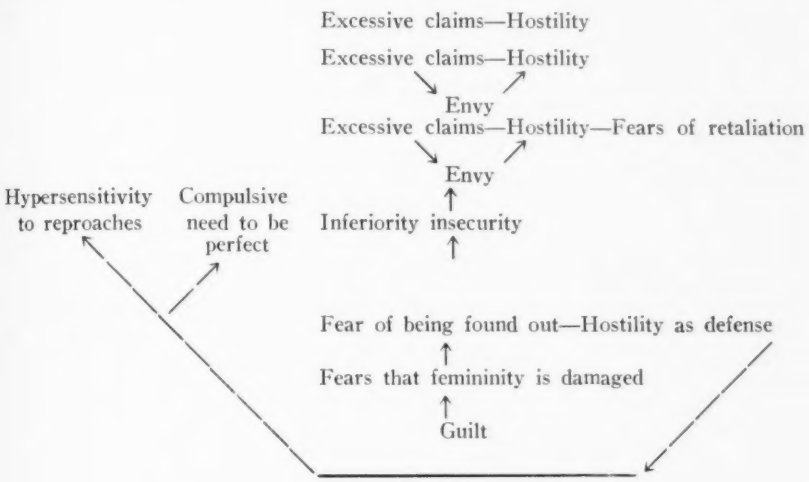
Owing to the insufficiency of the material, one may ask whether psychic suicide is possible.

THE CONCEPTS AND MISCONCEPTS OF THE ANALYTIC METHOD. DR. KAREN HORNEY (by invitation).

There is a basic misconception that there is no specific psychoanalytic method; that the analyst merely follows the patient's free associations and arbitrarily interprets the patient's behavior, chiefly in terms of early sexuality, with no safeguards against suggestion of thoughts alien to the patient. However, psycho-

analysis is gradually becoming systematized; it can be taught and is worthy of the name of a "method."

The method rests on the principle of free association, which implies the working hypothesis that there is a close psychic connection between ideas that are closely associated in time, even though there is no apparent logical connection between them. The data of psychoanalysis are of two kinds: (1) the statements of the patient, and (2) the direct observations of the patient's behavior by the analyst. The former vary in reliability because of possible distortions of facts and because of the patient's self-deception as to his emotions; the latter are rather exact. These two sets of data are used to complement each other. The statements of the patient help the analyst to understand attitudes which the patient exhibits. In complete analyses the interpretations start with and are based on the clearly observed attitudes because (1) the analyst can be certain only of these; (2) these surface attitudes are barriers inhibiting deeper insight, and (3) the consistent systematic analysis of the various attitudes in sequence reveals the causal emotional chains, leading from the most superficial to the deepest layers of the structure of the neurosis. This technic of analysis is outlined here.



DISCUSSION

DR. LOUIS CASAMAJOR: Dr. Stern has mentioned some philosophic considerations in the problem of the psychoneuroses in women. I often ask myself why most of our patients are women, and I refer not only to the patients of the neurologists and psychiatrists, but to those of every physician, except perhaps the genitourinary specialist. It is probably for the same reason that in an audience of this sort there is always a predominance of women. There must be something in the female personality and character which tends toward neurosis, and many theories have been presented to account for it. It is sometimes said that civilization may be responsible, especially in the case of those neurotic women who were children and adolescents in the mid-Victorian era, but that is pretty far back now. It was hoped that the "flapper" of the early twenties was not going to develop neuroses, but she seems predisposed to neurosis quite as much as her mother or her grandmother. Dr. Stern pointed out one thing which I think takes this problem out of the realm of social tradition and brings it down to something fundamental. He brings it down to the question of the fundamental feeling of inferiority of girls, owing to sexual inferiority because of the lack of a penis. Of course, in a little girl that refers merely to urination, and she feels that inferiority before she has any real consciousness of what genital sexuality means; her concern is mainly with

the definite organic inferiority in the urinary function. Then comes the attempt of the child to understand her own inferiority, usually complicated by the feminine preadolescent masturbation, of which there is no counterpart in boys. Later comes the trauma of menstruation, which the child has to meet during the adolescent period, with its bearing on the castration phenomenon, and finally the problem of adaptation to intercourse, again a rôle of more or less inferiority. These things start from a central core, as Dr. Horney has so well shown, and if I were to attempt to build up the subject I should start from the bottom of Dr. Horney's diagram. She has shown the breaking-down in the analysis.

Dr. Brill covered a good deal in the field of suicide, beginning with persons who really want to kill only a part of themselves. As Dr. Brill pointed out, that part belongs to the father. Those who have had the opportunity of seeing persons who have unsuccessfully attempted suicide have probably been impressed, as I have been, with the fact that none of them wants to die; even those who have shot themselves through the head, and who have severed the optic nerve and are completely blind still want to live. While the death wish undoubtedly plays some part, it is only a partial affair. The fundamental wish, as Dr. Brill pointed out, is really the wish to live. The psychic suicide, the wish-suicides, are extremely difficult to understand. I have in mind the case of an Indian coolie which was reported to me by a British medical officer. The coolie, while walking across a field, stepped on an iron hoop which flew up and hit him on the shin. He was convinced that he had been bitten by a cobra; no one could change that belief, and he started out to die. The surgeon put a couple of drops of croton oil in one of the man's eyes, which gave him so much to think about that he forgot to die. The death wish is undoubtedly more or less prominent, but I believe that in most people it has shifted into something else.

Dr. Horney has given an excellent picture of psychoanalytic procedure, much more of the philosophy of the procedure than of any particular technic. I was particularly grateful to her for laying more stress on the meaning and the aims of the analytic procedure than on any particular point of technic. The diagram showing the levels at which various complexes occur is extremely accurate. I have never seen a neurosis more accurately portrayed in a graphic way.

Dr. Marion E. Kenworthy: I have two brief notes in regard to Dr. Stern's paper: A boy, aged 3, who was given the privilege of seeing his baby sister bathed for the first time when she returned from the hospital, remarked to his mother: "What a terrible cut!" Also, a girl, aged 2½, whose baby brother and mother returned from the hospital, showed intense anxiety for about three days after their return. She would not eat or sleep. When the mother consulted me, I suggested that she review some of the things which were new in the child's recent experience, besides the fact of her return from the hospital with the brother. The mother reconstructed the picture; she had read that it was an excellent idea for girls to see their baby brothers bathed. This had happened on the first morning after their return. I suggested that she talk the matter over with the girl and ask her how she felt about helping her mother bathe the baby. The child's reaction to the question was: "Why didn't you make me fancy like you made my brother?"

Dr. Brill's paper is interesting; any one in the analytic field can add many other interesting examples. There is one aspect of it which extends beyond this so-called psychiatric principle. One of my students, a priest who has a parish, reported the case of a patient who had attempted suicide two or three times. "She was so filled with the desire to die," he said, "but I have kept her alive; she comes to confession once a week and shows such a profound sense of guilt that I give her a penance to perform, and the heavier penance I impose on her, the happier she is during the following week."

It is always a privilege to hear one of Dr. Horney's brilliant papers. In recent years much has been published on methods of psychoanalysis. Too much has been selected for presentation to the public without due appreciation as to reliability of content. Psychoanalysis opened a new and ever-increasing field of

observation and research. In its true clinical aspects, the method of approach to personality problems through investigation of the emotional or psychologic processes of each person's life experience furnishes the well equipped analyst with an opportunity to see the whole picture of cause and effect relationship in the patient's illness.

Obviously, as Dr. Horney has so ably pointed out, the statement of the patient alone is bound to vary in its reliability, because of the possible distortions of facts and because of the patient's self-deception as to his emotions. It does not matter what happens to one, but how one feels about what happens. This continues to be an important factor in human experience. Dr. Horney's emphasis on the importance of observation of the patient's behavior by the analyst cannot be repeated frequently enough.

Training of young analysts in this more organized and basic method of approach to patients' problems assures the patient a more adequate therapy.

PHILADELPHIA NEUROLOGICAL SOCIETY

Jan. 26, 1934

A. C. BUCKLEY, M.D., *President, in the Chair*

A CEREBRAL TUMOR WHICH PRODUCED TRANSIENT UNILATERAL EXOPHTHALMOS DEPENDENT ON THE POSTURE OF THE HEAD. DR. W. B. CADWALADER and DR. D. B. CONNERY.

History.—A white man, aged 55, who was admitted to the Presbyterian Hospital on Dec. 26, 1933, had complained of occipital headache for two years. He was operated on for disease of the right ethmoid and sphenoid sinuses two years before admission. Eighteen months before admission right mastoidectomy was performed. The headache became more severe during July and August 1933. The patient was free from pain from then until late in September 1933, when the headaches returned with greater severity. Impairment of memory and confusion seemed sufficient to indicate to the family some mental deterioration, yet the patient held his position as a statistician until November 1933. He leaned to the right when walking, and was very unstable, swaying and running forward. This was interpreted as festination. The right ear had been draining mucopurulent material for twenty years.

Physical Examination.—The patient was drowsy and confused. He went to sleep during the examination and even while being catheterized. He slept most of the day, as well as at night. This was interpreted as hypersomnia, a symptom of cerebral disease. The reaction of the pupils to light was sluggish, and no changes were detected in the eyegrounds. There was profound left facial palsy of the central type. All tendon reflexes were normal and equal. There was no weakness of any extremity. Rectal examination disclosed third degree hypertrophy of the prostate. Pus was present in the urine; this made it difficult to determine whether the mental condition was due to the effects of infection, or was actually hypersomnia of cerebral disease.

Course.—On Jan. 1, 1934, there developed marked exophthalmos on the right side. At the time the patient was lying on the left side, with the head rotated to the left, in deep coma. The exophthalmos developed in about three hours. There was no pulsation of the orbit, and no bruit was present. The blood pressure at this time was 175 systolic and 125 diastolic. The temperature was 101 F., the pulse rate, 60, and the leukocyte count, 15,650, with 94 per cent polymorphonuclears. When the patient was placed flat on the back for about an hour the exophthalmos decreased slightly but noticeably. Later, he was turned on the right side, with the head rotated to the right. The exophthalmos continued to decrease and by the

next morning, Jan. 2, had disappeared. The blood pressure was 140 systolic and 90 diastolic, and the pulse rate, 84. The eyegrounds showed no change. The condition was diagnosed as tumor involving the facial area and extending to the right temporal region. Operation was considered, but owing to the patient's genito-urinary infection it was not attempted. The patient died at 9:09 p. m. on January 2.

Postmortem Examination.—A tumor, 3 cm. in diameter, was attached to the anterior surface of the inferior portion of the right temporal lobe. It appeared to arise from the dura mater covering the greater wing of the right sphenoid bone, and was just adjacent to the right optic nerve and the internal carotid artery. Section of the brain showed constriction of the right lateral ventricle. Purulent material was found in the right ethmoid sinus and the right mastoid cells.

Microscopically, the tumor appeared to be a fibroblastoma.

Comment.—The cause of the left facial palsy is obvious. We have attempted to explain the transient exophthalmos on the right side by assuming that with the head rotated to the left the tumor moved toward the midline, pressed on the adjacent tissues and impeded the return of the venous blood from the orbit. With the head rotated to the right the tumor moved away from the midline, the pressure was removed and the exophthalmos gradually disappeared.

FOOT DROP WITH SIMILAR AND ASSOCIATED SYMPTOMS: REPORT OF THREE CASES.

DR. SAMUEL B. HADDEN, DR. MACKINNON ELLIS and DR. JOSEPH FITZMAURICE.

The three cases of foot drop reported here have common factors which are of considerable interest and, we believe, infrequently noted.

CASE 1.—Mrs. A. C., a white woman, aged 51, had fibrillation during an attack of cardiac decompensation in 1930. Since, she has shown signs of decompensation on several occasions. On the night of Jan. 13, 1932, she sat with the right leg crossed over the left at a theater. After a short time in this position she began to experience numbness and then pain in the right foot and leg. On uncrossing the legs the numbness increased, and shortly after the pain became severe in the foot and the leg below the knee. She was forced to leave the theater because of the discomfort, and two injections of morphine, ½ grain (0.032 Gm.), were necessary for relief during the night.

At the first examination a complete foot drop was noted. On the morning following the onset a purplish discolored area was noted on the anterolateral aspect of the leg from about 2 inches (5 cm.) below the knee to about the junction of the lower and middle thirds of the tibia. Anesthesia to all forms of sensation was present in this area and extended for some distance over the surrounding healthy skin. Gangrene developed in the area noted, and in ensuing weeks a slough formed with ultimate exposure of both bones. Osteomyelitis developed, and several sequestrums were removed. After several months, granulations appeared and the deep wound began to fill in. At present, the ulcer has filled in and is covered with epithelium except over a small sinus through which an occasional small piece of necrotic bone is discharged. Dorsiflexion of the foot is now surprisingly good, about 50 or 60 per cent as powerful as that of the healthy foot, and the patient is able to walk about with a slight limp, the result of pain and slight foot drop; sensation is markedly diminished over the healing area.

In August 1932, the patient had a cerebral vascular disturbance which produced a transient left hemianopia.

CASE 2.—W. H., a white man, aged 38, a park guard, had been under observation for four years because of auricular fibrillation with frequent periods of cardiac decompensation. The previous medical history was without significance except for frequent nosebleed in the past ten years and scarlet fever at the age of 12. In the early part of April 1932, symptoms of decompensation gradually developed, but the patient continued to be ambulatory until April 10, 1932. A few days before entrance to the hospital he experienced a sudden stabbing pain

at the base of the right thumb, which radiated into the thumb. The pain was intense for about forty-eight hours but gradually subsided, leaving numbness and objective and subjective coldness of the whole thumb.

On April 10, he was confined to bed in a state of severe decompensation. On the night of May 17 he awakened at about 10 p. m. with severe pain in the right leg below the knee. Morphine was required for relief from pain. Early in the morning of the following day he was awakened from sleep by a similar excruciating pain in the left leg. Because of dyspnea he was propped up in bed with a bolster beneath the knees.

Neurologic examination on May 18 revealed complete bilateral foot drop with preservation of other movements in both lower extremities. On the anterolateral aspect of the right leg was an area of pale purplish discoloration extending from about 2 inches below the knee down to about the junction of the middle and lower thirds of the tibia, over which the skin temperature was markedly reduced. On the left leg in the same area the skin temperature was markedly reduced, but no discoloration was noted. In both these areas sensations to pain, touch, heat and cold were lost. Neither dorsalis pedis artery could be felt to pulsate. The outer side of the dorsum of each foot was hypesthetic to all forms of sensation. Within forty-eight hours of the onset dry gangrene developed in both these areas, and about five days later, associated with great aggravation of pain, both legs became entirely gangrenous below the knees. The patient died on May 29.

CASE 3.—G. W., a white man, aged 39, was admitted to the neurologic service of the Episcopal Hospital on Oct. 31, 1933, and discharged on December 5. He had been well until about the second week in August 1933, when on returning from work he went to the toilet to have a bowel movement. He remained on the commode reading a newspaper for about a half hour, and on getting up fell to the floor because of weakness and numbness in both feet and legs. He went to bed shortly after, but the numbness and tingling continued. On the following morning both legs and feet felt "numb" and "dead" and were very weak and slightly swollen; on the inner aspect of the right great toe a bleb filled with blood serum was noted. This bleb later became dry, and at the time of admission was a typical gangrenous ulcer. From the time of onset until admission the patient was unable to walk without assistance and spent most of his time in bed or in a chair.

On admission to the hospital there was slight edema of both legs below the knees. The anterolateral aspect of each leg was perceptibly colder than other portions of the extremities. There was definite, not complete, foot drop on both sides, and the patient was unable to walk. A gangrenous ulcer was noted on the inner aspect of the right great toe. Position and vibratory sensation were normal. Below a line at the tibial tubercle of the right leg sensation to pain was diminished, and it was entirely absent over the anterolateral aspect of the leg and the dorsum of the foot. In the left leg the findings were the same except that pain was normally perceived over the dorsum of the foot. Sensation to heat and cold was absent in the same areas in which sensation to pain was lost. Other phases of neurologic examination gave normal results. Pulsations in the dorsalis pedis artery were not detected in either foot.

Medical examination in this case revealed heart sounds of poor quality, with systolic blood pressure of 110 mm. and diastolic pressure of 75 mm. Electrocardiograms showed left bundle branch block with definite myocardial depression, a simple bradycardia and sinus arrhythmia.

Under tonic treatment and baking of the extremities the patient improved slowly, and on leaving the hospital he walked with a typical steppage gait. He still has partial bilateral foot drop, lowered skin temperature and hypesthesia to all forms of sensation over the anterolateral aspect of the lower portion of each leg and a slowly healing ulcer of the right great toe. The gait still shows steppage characteristics. Pulsations of the dorsalis pedis arteries cannot be noted.

Comment.—In all three cases there is a history of pressure over the popliteal vessels: In case 1, the pressure was due to crossing the legs; in case 2, to a bolster used beneath the knees for support and in case 3, to pressure of the

rim of the toilet seat. Each patient had severe cardiac disease with resulting sluggish circulation. We believe that complete or partial thrombosis of the anterior tibial vessel occurred in each case, and that the resulting ischemia and edema of the anterior tibial nerve caused the foot drop. In the first two cases definite gangrene with sloughing occurred in the area of supply of the anterior tibial artery. In case 3 the gangrenous ulcer of the great toe was probably the result of thrombosis of a terminal branch from the dorsalis pedis artery. In this case there was no gangrene in the main area of supply of the anterior tibial artery, but circulatory and secondary sensory impairment in this area was unmistakable.

We do not wish to imply that all cases of foot drop which are attributed to pressure on the nerve by crossing the legs are the result of thrombosis of the anterior tibial artery, but we wish to point out that a crossed leg position in which there is pressure over the anterior tibial nerve in its superficial position is not one of great comfort. In persons with impaired circulation, we believe that pressure over the popliteal vessels with resulting thrombosis of the anterior tibial branch may be an important factor in the development of foot drop.

DISCUSSION

DR. J. W. MCCONNELL: Several years ago I had under my care a physician with paralysis of the right leg, involving the peroneal distribution, and also a gangrenous condition of the fourth and fifth toes of the right foot. The man had suffered from severe cardiac and arterial disease for several years, although at the time of death he was still young. The toes were removed by a necessary operation. The paralysis persisted until the time of death from lobar pneumonia, five months later.

The diagnosis of the causative condition in his case was obliterating endarteritis.

TRANSVERSE LESIONS OF THE SPINAL CORD. DR. FREDERIC H. LEAVITT.

Cases presenting symptoms of transverse lesions of the spinal cord are relatively frequent in hospital practice, and not infrequent in private practice. In this series of fifteen consecutive cases one is struck by the variation in the nature of the causative pathologic condition. In case 1, purulent myelitis; in cases 2 and 3, trauma; in cases 4, 5 and 6, a metastatic carcinoma of the cord; in case 7, a giant cell tumor of the vertebrae; in case 8, tuberculous spondylitis and crushing of the cord from osteopathic manipulation for relief from the pain in the neck; in case 9, a congenital lipomatous tumor of the cord in a baby, presumably having developed prior to birth; in cases 10, 11 and 12, rupture of the cervical cord due to too vigorous manipulation of the infant in relation to the after-coming head in cases of breech presentation; in cases 13 and 14 (almost consecutive instances), a myeloma of the vertebrae producing complete transverse myelitis; case 15 was the only one of the series in which the patient recovered. This patient had syphilitic meningomyelitis of the cord that was cured by an attack of influenza, associated with high fever, of several days' duration.

CASE 1.—A married man, aged 21, was lifting bags of coal, weighing about 100 pounds (45 Kg.), when he suddenly felt pain in the middorsal region of the back. The pain continued; one week later there slowly developed transverse myelitis of the cord in the middorsal region coincident with an elevation of temperature to from 101 to 105 F. and motor and sensory paralysis below the nipple line on both sides. The spinal fluid was yellow, was under a pressure of 20 mm. of mercury, contained 8,800 cells and showed complete coagulation on standing. Roentgenograms showed no evidence of pathologic changes in the spine. Laminectomy revealed what appeared to be necrosis of the cord with a burrowing abscess on both sides of the spine in the muscles of the erector spinae group. There appeared first ataxia in the lower extremities; in a week there developed complete paraplegia with paralysis of the bowel and bladder. Repeated cultures revealed Staphylococcus aureus. The patient died six weeks after the onset. At autopsy, the wound made at laminectomy was found to be purulent, and pus was seen in

the spinal canal, there being a large amount between the meninges and the cord. The cord showed what appeared to be transverse myelitis at the level of the seventh dorsal vertebra, and there was pus between the dura and the arachnoid as well as between the arachnoid and the pia. For a distance of 2 cm. on each side of the thoracic segment the cord was reduced to a mere shell filled with creamy, purulent material. Below this level the central portions of the cord were necrotic, although the main features of the cord were still discernible; this pathologic condition was present to the end of the cord. Above the level of the seventh thoracic segment the central canal was dilated and plugged with thick pus as far as the lower cervical segment. There was a thin layer of pus over the base of the brain and some pus in the ventricles. The pathologic diagnosis was purulent myelitis of the spinal cord at the seventh thoracic segment and below and secondary purulent cerebrospinal meningitis.

CASE 2.—A woman, aged 31, had been in good health until she was in an automobile accident, which was followed by complete paralysis from the neck down. A roentgenogram revealed dislocation of the sixth cervical vertebra. Immediate laminectomy revealed the cord to be crushed in the region of the second and third cervical vertebrae.

CASE 3.—A lad, aged 16, had been well until he was in an automobile accident, which was followed by complete paralysis from the neck down. A roentgenogram revealed no abnormality in the vertebrae. The patient died on the fourth day. Autopsy revealed hemorrhage into the spinal cord, with crushing of the cord from luxation of the second and third cervical vertebrae and apparent spontaneous reduction of the dislocation.

CASE 4.—A man, aged 68, had a progressive transverse myelitis of the cord in the lower dorsal region which developed within two months. A roentgenogram revealed metastatic malignant disease in the lower part of the spine and the pelvic bones. A prostatectomy had been done four years previously for carcinoma. Autopsy revealed multiple metastases in the bones of the pelvis and vertebrae, producing malignant compression of the cord at the level of the eleventh dorsal vertebra.

CASE 5.—A woman, aged 39, for six months prior to death had noticed gradual loss of power in the lower extremities, which increased to a complete transverse myelitis below the eleventh dorsal vertebra. Examination of the spinal fluid revealed a marked increase of white cells and evidence of spinal "block." Cystoscopic examination revealed primary carcinoma of the wall of the bladder. A roentgenogram of the spine revealed no apparent metastasis in the spine. Autopsy showed carcinomatous metastasis in the spine at the level of the eleventh dorsal vertebra, with complete crushing of the cord.

CASE 6.—A man, aged 70, had been well until six weeks prior to death. Within a week of the onset there appeared rapidly increasing ataxia of the legs, which was soon accompanied by pain radiating around the chest just below the shoulders. The condition increased so rapidly that within one month from the onset the patient was bedridden and had total motor and sensory paralysis from the nipple line down, together with paralysis of the bowel and bladder, abolition of all tendon reflexes and deep trophic sores on the pressure points of the back. Injection of iodized poppy-seed oil 40 per cent into the spinal canal during the second week of the illness showed a partial block at about the first dorsal segment. Roentgenograms of the spine and chest at first revealed nothing abnormal, but finally showed a carcinoma of the apex of the left lung, with contiguous involvement of the first dorsal vertebra and malignant encroachment on the spinal cord.

CASE 7.—A man, aged 19, was believed, in 1924, to have tuberculous Pott's disease of the lower dorsal vertebrae. An Albee operation was done at that time, the bone graft being obtained from the patient's left tibia. Following the operation pronounced scoliosis of the spine developed in the middle dorsolumbar region. Two weeks later the patient began to suffer from pain in both legs, radiating especially down to the left knee. A few days later he lost full voluntary control

of the left leg, and in a few weeks, control of the right leg. With the motor paralysis the pain in the legs disappeared, and a coincident paralysis of the bowels and bladder developed. A roentgenogram showed a regular, circular mass, slightly larger than the body of the eleventh and twelfth thoracic lumbar vertebrae, at the lower level of the Albee bone graft. The appearance was suggestive of an osteogenic sarcoma. Injections of air into the spine showed complete block at the level of the first lumbar vertebra, no air having reached the brain. The Queckenstedt test also demonstrated complete block in the spinal canal. Operation revealed a bony mass invading the spinal canal at the level of the Albee bone block, and a portion removed for diagnosis was found to be "primary giant cell tumor." The patient lived four months at home with complete transverse myelitis of the cord, complete motor and sensory paralysis of the legs, paralysis of the bowel and bladder and large trophic bed sores.

Case 8.—A man, aged 30, for some weeks prior to the onset of the illness had complained of pain and aching in the neck. He was advised to have osteopathic treatment. During the procedure he had a sudden severe pain in the neck and almost instantly became paralyzed from the neck down. On admission to the hospital on the next day he exhibited flaccid paralysis of both arms and spastic paralysis of both legs, definite bilateral ankle clonus and a bilateral Babinski sign, and paralysis of the bowel and bladder. A roentgenogram showed tuberculous destruction of the sixth cervical vertebra. The patient remained in the hospital for two months, when an Albee bone graft operation was performed, following which he improved so much that in five months he was able to use both legs and both arms; much of the spasticity had disappeared. Nine months after the bone graft operation the patient died of generalized tuberculosis.

Case 9.—An infant, aged 11 months, was admitted to the Children's Hospital with the history that since birth she had had an incomplete flaccid paralysis of both arms, with marked spastic paralysis of both legs, some palsy of the chest and abdominal muscles and apparent lack of control of the sphincters of the bowel and bladder. The head was held rigidly on the neck, and the patient cried when any attempt was made to move the head. As this condition had existed from birth, a tentative diagnosis of bilateral brachial birth palsy was made. The spinal fluid was yellow and under a pressure of 20 mm.; the Wassermann reaction was negative and the Queckenstedt test showed a partial block. A roentgenogram of the spinal column revealed no abnormalities. There was no apparent increase of the signs of complete transverse lesion of the spinal cord in the lower cervical region. Within four weeks of admission the patient died of bronchopneumonia. Autopsy revealed a lipoma of the cervical portion of the cord, extending from the fifth cervical to the first dorsal vertebra, completely filling the canal, intracordal in position and reducing the cord to a mere ribbon of tissue for a distance of about 2 inches (5 cm.).

Cases 10, 11 and 12.—These three cases from the Children's Hospital were in infants born by breech presentation; immediately following birth they were found to have complete transverse lesions of the cervical cord, typified by flaccid paralysis of both arms, spastic paralysis of both legs and paralysis of the muscles of the chest and abdomen, giving the characteristic picture of marked ballooning of the abdomen and marked retraction of the wall of the chest. Breathing was entirely diaphragmatic, and the cry was scarcely audible. There was paralysis of the sphincters of the bowels and bladder. Autopsy in each case revealed rupture of the cord in the lower cervical region, the cord having been reduced to a mere thread surrounded by hemorrhage. The cases were due to traumatic rupture of the cord during breech delivery.

Case 13.—The patient had been well until three weeks prior to admission to the Reading Hospital, when he noticed a sudden sharp pain in the middorsal region of the back while lifting a piece of furniture weighing about 100 pounds (45 Kg.). For three weeks no symptoms other than pain were present; then, within twenty-four hours, there developed signs of a transverse myelitis of the

cord in the middorsal region. The temperature rose to from 101 to 103 F. The spinal fluid was entirely normal. The roentgen diagnosis was "compression fracture of the twelfth thoracic vertebra." Within a week the patient was completely paralyzed below the middorsal region, both as to motor power and as to sensory perception, and there were paralysis of the bowel and bladder, trophic bed sores on the back and an area of painful hyperalgesia completely around the chest at the ensiform area. Laminectomy revealed no pathologic changes. The patient died six weeks after the onset of symptoms. Autopsy revealed the ninth, tenth and eleventh vertebrae to be "soft and carious." There was a purulent discharge in the spinal canal, and the spinal cord was completely destroyed distally to the twelfth thoracic vertebra. At the level of the twelfth vertebra on the anterior surface of the spine, there was a large tumor mass. This growth appeared to arise from the intervertebral disk of the twelfth thoracic vertebra. It was composed of grayish material of uniform structure, and it "flowed out" about the spinal column, forming a "collar" about it for a width of about 2 cc. It was noticed to project into the spinal canal, causing compression of the cord from before backward. The vertebra was necrotic and wedge-shaped. Microscopically, the tumor was composed of oval cells possessing an abundant, intensely acidophilic, rather opaque cytoplasm and eccentric round or slightly oval, deeply stained nuclei. These cells were arranged in no regular pattern, but existed in solid masses, with a fine fibrous stroma. In some places there were open spaces surrounded by these cells; in others they appeared in broad, solid sheets. A good many fine capillaries and small, thin-walled venules traversed the growth. The cells varied greatly in size, producing a malignant picture. Some were very large and possessed two irregular hyperchromatic nuclei. Mitotic figures and nuclear forms suggesting amitotic division were found. In sections of the twelfth thoracic vertebra this tumor tissue was seen replacing the bone marrow. Dr. C. F. Geschickter, of Baltimore, diagnosed the tumor as "multiple myeloma."

Case 14.—For three months prior to admission to the hospital, the patient, a physician of 63, seen in the Reading Hospital, complained of pain in the mediastinal region. Being a physician, he was of the opinion that he was suffering with angina pectoris. A cardiac specialist was consulted, and a diagnosis of coronary disease was made. Despite treatment, the pain in the chest increased markedly in severity, and a week prior to admission to the hospital there appeared ataxia in the lower extremities. Examination showed abolition of the reflexes in the lower extremities, marked incoordination of gait and station and painful hyperalgesia at the nipple level. Within a week the picture had changed to that of a complete transverse lesion of the cord at that area, with the production of motor and sensory paralysis below the nipple level, loss of control of the sphincters of the bowel and bladder and trophic bed sores. The temperature remained normal until the last two weeks of life. Gastric analysis gave normal results. When the paralysis had become complete, the Queckenstedt test showed complete block of the canal; the fluid contained 84 lymphocytes, had a yellow color and gave a negative Wassermann reaction. A roentgenogram of the spinal column showed no abnormalities. Three weeks after the symptoms referable to the cord began the patient died. Autopsy showed the spinal cord to be normal above the fourth thoracic segment; below that level there were signs of pressure on the cord and the cord was very soft. Section revealed the entire center of the lower portion of the cord to be completely necrotic. The outer layers were preserved and served as a sheath for the necrotic center. A tumor growth was found bulging out between the bodies of the fourth and fifth thoracic vertebrae and appearing to arise in the intervertebral cartilage. It consisted of firm, grayish material of homogeneous color and consistency throughout. This growth appeared to form a "collar" about the spinal column at the fifth thoracic segment. Examination of the liver showed all the portal zones to be densely infiltrated with oval cells of various sizes, similar to those in the spinal tumor. The spleen was engorged with tumor cells of various sizes similar to those of the spinal growth. The distribution of these cells resembled that of leukemic infiltration. Numerous small

tumors in the dura of the brain were composed of the same cells as the spinal tumor. The tumor in the spinal cord was composed of oval cells of widely varying size packed into the interstices of a scanty hyaline matrix. The cells possessed an abundant, deeply acidophilic cytoplasm and large eccentric nuclei. The nuclei were round or slightly oval and stained deeply. They possessed a distinct "cart wheel" chromatin. There were a good many large cells which possessed several huge, hyperchromatic nuclei. This tissue pushed into and about adjacent muscle and nerves in a perfectly unhampered fashion, as if simply poured in. This tissue was examined by Dr. C. F. Geschickter who made a diagnosis of "multiple myeloma."

CASE 15.—A woman, aged 36, had motor and sensory paralysis below the level of the umbilicus, which had developed gradually during two months prior to admission to the Orthopedic Hospital. On admission she had complete motor paralysis of both legs, paralysis of the bowel and bladder, deep sloughing bed sores over the sacrum, trochanters and heels, and sensory paralysis up to the level of the umbilicus. The Wassermann reaction of the spinal fluid was strongly positive; the cell count was 200 lymphocytes, and the colloidal gold test revealed a syphilitic curve. The case was diagnosed as "gumma of the cord" or "syphilitic meningomyelitis." The patient did not respond to any form of antisyphilitic treatment and was sent home with the expectation that she would die. One year later she reported at the hospital, walking and apparently well, with the statement that recovery had begun following an acute attack of influenza during which she had a high fever for two weeks. Since, she had made a rapid and uneventful improvement to complete recovery.

Summary.—1. Among the fifteen cases presented, there were nine distinct pathologic entities as causes of the syndrome. 2. Roentgen examination failed to help in differential diagnosis. 3. The greatest aid in establishing a differential diagnosis was afforded by lumbar spinal tap. 4. "Posterior cord ataxia" was frequently the initial symptom of invasion of the cord. 5. The danger of physical therapeutic manipulation of the spine in unsuspected cases of tuberculous spondylitis is illustrated. 6. "Cure" in a case of specific myelitis by an intercurrent febrile infection is recorded. 7. The seventh dorsal vertebra as a point of greatest weakness in the spinal arch is emphasized. 8. A "primary giant cell" tumor developing at the site of an Albee bone graft is recorded. 9. An intramedullary lipoma developing and producing symptoms prenatally is recorded. 10. The relative frequency with which rupture of the cervical portion of the spine occurs in breech presentations is emphasized.

DISCUSSION

DR. W. B. CADWALADER: Dr. Leavitt has described a case in which excessive muscular exertion, as in lifting, preceded the rapid onset of paraplegia, with sensory changes throughout the lower limbs and trunk up to the level of the lesion. At operation, pus was found in the spinal epidural space. I have recently seen two cases of this kind and have been impressed with the regularity of the history and mode of onset of the symptoms. The condition is due to an infection, generally with staphylococci secondary to long-standing furunculosis or other suppurative process. In my cases, furunculosis preceded the onset of paraplegia. Dr. Leavitt has not mentioned whether or not this was observed in his case. Severe root pain and localized tenderness of the spine, aggravated by coughing or straining, although not observed in every case are among the early symptoms. They frequently precede the onset of paralysis. Complete or nearly complete paraplegia with paralysis of the bladder and rectum generally develop rapidly, that is to say, within twenty-four hours or within a few days, but may not appear until later. Cutaneous sensation is severely affected up to the level of the lesion. Complete or partial spinal block can usually be demonstrated. The character of the lesion should be suspected with the history of previous suppuration and the rapidity of the onset of paraplegia. Most of the cases recorded in the literature developed in this manner, associated with or preceded by long-standing furunculosis. Different

names have been used to describe the condition. I have used the term "circumscribed extradural intraspinal nontuberculous abscess." Circumscribed subdural abscess is much more rare. When it does occur, however, it is associated with diffuse suppurating spinal meningitis. Bearing in mind these facts, a clinical diagnosis can generally be made without great difficulty.

DR. N. W. WINKELMAN: Dandy has been able to show anatomically that most of the cases of epidural abscess are located at approximately the same level, owing to peculiarities of lymphatic drainage into the epidural space at the sixth thoracic segment. Dr. Cadwalader's observations that these cases are alike clinically would be borne out by these facts.

ACUTE NONEPIDEMIC DISEASE OF THE SPINAL CORD. DR. A. C. BUCKLEY.

This article will be published in full elsewhere.

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HENRY ALSOP RILEY, M.D., *President, in the Chair*

THE MECHANISTIC APPROACH TO THE PROBLEM OF MENTAL DISORDER. DR. MILTON HARRINGTON, Napanoch, N. Y.

In attacking the problem of mental disorder there are two fundamentally different kinds of psychology between which one may choose. The first is the psychology of the psychoanalyst. Psychoanalysis is an attempt to explain mental disorder in terms of motive or desire. According to psychoanalytic theory, psychopathic phenomena are manifestations of impulses or desires which are bottled up in the unconscious and tend to manifest themselves in disguised or distorted forms; by psychoanalysis it is claimed that these unconscious desires may be brought to light. This kind of psychology, in which everything is explained by attributing it to a motive or a desire, one may call motivistic psychology. In building his motivistic psychology the psychoanalyst makes use of concepts which are essentially animistic.

Instead of using animistic concepts, which have been handed down from a prescientific age, one may think of behavior as due to the action of a complex mechanism composed of sense organs, nervous system, muscles and glands. Abnormal thoughts, feelings and actions may be attributed to the limitations and defects of this mechanism, by reason of which it not infrequently fails to respond in an adequate way to the demands made on it. This kind of psychology, which would explain abnormal behavior as due to faulty action of the anatomic mechanism by which all behavior, both normal and abnormal, is produced, one may call mechanistic psychology.

Looking at the matter from the mechanistic point of view, behavior is seen as a never-ending series of reactions to the situations of life; these responses occur as a result of the stimuli provided by the situations. This is just as true of forms of behavior which do not meet the needs of the environment in which they occur, and which are called psychopathic, as it is of those which are called normal. The nature of a reaction depends in every case on two factors: (1) the nature of the stimulus acting on the organism and (2) the nature of the reacting organism. The latter in turn depends on three factors: (1) heredity, (2) education, by which is meant the sum total of the impressions stamped on the brain as a result of the experiences to which in the course of his lifetime the individual person is subjected, and (3) injury or disease. Altogether, therefore, there are four variables which are the determining factors in human behavior: (1) heredity,

(2) education, (3) somatic disease and (4) environmental stimuli. The causes of all forms of mental abnormality are to be found in these four factors. These conclusions, to which the mechanistic theory leads, are different from those of the psychoanalyst, who passes over these factors as too simple and obvious for serious consideration, focusing attention instead on new and strange causes disclosed by him.

As the mechanistic theory leads to different conclusions in regard to etiology, so also does it lead to different conclusions as to treatment. Since, according to psychoanalytic theory, mental disorder is due to wishes which are productive of harm because they are unconscious, the thing to do to effect a cure in any case is to bring the trouble-making wish into consciousness; this is what the psychoanalyst claims to do in the course of analysis. But what of mental hygiene, the art of right living, as a way to mental health? Right living as a way to mental health can have little significance for the psychoanalyst because, according to his theory, mental disorder is due not to wrong living but to unconscious, perverted sexual desires which have been present from infancy and can be removed only by psychoanalytic treatment.

Looking at the matter from the mechanistic point of view, however, since mental ills are due to bad heredity, faulty education, somatic disease or difficult or unwholesome situations, the preservation of mental health involves consideration of these four factors. It is a matter (1) of breeding a race of potentially healthy and efficient people, (2) of giving these people the kind of education or training which will make the most of their potentialities, (3) of caring for their bodily health and (4) of giving them the environment best suited to their requirements. In short, it is a matter of creating a wholesome environment and of living according to the laws of health within that environment.

DISCUSSION

DR. FOSTER KENNEDY: Dr. Harrington has made a plea for the consideration of the human animal as a whole. Physiologists often speak of the cerebral cortex acting as a whole. I have never been able to understand clearly what they mean. Head used the phrase in his writings. I have always thought it a confusing term, but I think I understand what is meant by a human being acting as a whole, because the metazoic, multicellular animal functions in each act as unifiedly as does the unicellular animal. Each act of behavior is the resultant of the forces that are in the organism, so that I think one may properly speak of the human being as acting as a whole.

Dr. Harrington has spoken rather adversely of Freud's theory as being a purely motivistic conception of human behavior. If one speaks decidedly on any side in this rather amorphous world one gets the reputation of being prejudiced. One's antagonism is only a criticism of psychoanalysis. It is one angle of view only. Mind symbolizes everything one sees. Every truth has many aspects; one sees but one aspect at a time and that first and clearest which appeals to the consciousness of the observer. The important thing is to try to have more than one angle of vision; my feeling is that there is a certain belief among psychoanalysts that psychoanalysis has absorbed psychiatry. They believe that they are psychiatrists and that unless one is a psychoanalyst one is not a psychiatrist, and that he who is not for them is against them. They suggest that one has no right to have a notion or an idea of human personality in other terms. Voltaire, Shakespeare and George Moore were not psychoanalysts, but I venture to say that they knew more of the human spirit and its motivistic phenomena than most of Freud's disciples. The great novelist is perhaps the greatest of all psychiatrists. He understands perhaps better than the psychiatrist knows.

I believe that the freudian contribution is this: Freud has demonstrated that there is a phylogeny of personality. By that, I mean that every one is a microcosm of the race. From the egg to death one passes through stages in the body which are at least similar to those through which the whole race has passed. Freud has made it clear that in emotion, in striving, in the preponderance of one instinct

over another at different parts of one's life, one has a like phylogeny of personality. The child is a savage; its sexual instincts emerge by gradual progression from preoccupation with one orifice to preoccupation with another. That is a real contribution to knowledge. Freud himself has lately said that he is not sure whether it is a contribution to therapeutics, but at least it is made clear how instincts develop inside the microcosm of each man's body. This analysis of behavior has been a great compensation against the materialism and dogma of the "cellular" epoch which naturally evolved in the nineteenth century; but if one is to think solely in terms of this reversion to dualism, this ecclesiastical view of man, which has beset minds and lamed thoughts for 2,000 years, if one is to think that such patterning and docketing is the whole of psychiatry, then one is sinning against the holy ghost of medicine. The plain facts of fever psychosis and glycosuria in shell-shocked soldiers must be remembered. If one imagines each human being to be a single unwavering, unchanging uniformity against stimuli which are directed, and if one postulates that every person invariably reacts to stimuli in the same way as every other person, one is saying something contrary to human experience and common sense. All persons vary as much in their personalities and reactions as in their faces. They differ, as even Freud has stated, in their infantile reactions. But what he has not laid stress on is that they differ in their inherited endowment. I am not sure whether "inherited endowment" is an absolute concept. I am not sure that the dogma that there is no such thing as the inheritance of acquired characteristics is true. Indeed, part of the trouble in this whole discussion is that one looks at the matter narrowly and thinks in terms of too small units of time. One does not see the results of inheritance of acquired characteristics because of the paltry shortness of life, and since one does not see them the mind's eye fails to grasp them. There is a good deal of evidence in the animal world that such transmission of acquired characteristics exists, that a defective germinal cell will produce a defective descendant and that the defective descendant will procreate a defect in its descendant. It has been shown by the injection of pulverized lens tissue of rabbits into fowls that their serum thereafter put into pregnant rabbits produced creatures with defective eyes which are inherited for eight generations. If that can happen in lower animals, it may happen in man; but the human nervous system is so complex and so much more evolved that the matter cannot be appreciated in one generation. Inheritance and the care of the stock are perhaps the most important factors in the prevention of the psychotic state. There are the results of great injury and great shock; but consider, for example, the greatest disasters and emotions, especially of war. The statistics of the last war, which was a great laboratory for this investigation, show that while psychoneuroses were very frequent—in the tens of thousands—there was no increase in psychoses in the troops as compared with the same number of people in civil life. If psychoses are produced only by "biologic pressure," how can one account for the absence of an epidemic of psychoses in war? One cannot. Of course, the pressure of the herd instinct and the pressure of sex and of hunger impinge on, mold, modify and direct the growing organism. A man who may be potentially homosexual may be made completely homosexual by experience, but he must be nearly a complete homosexual, I am sure, not to resist the normal trend of physiologic living.

I shall criticize only Dr. Harrington's use of the phrase "mechanistic approach." I do not think that he is using or advocating a mechanistic approach. What he is advocating is that one should employ all one's physiologic, anatomic, social and anthropologic knowledge and all one's bacteriologic, histologic and morphologic knowledge, bringing them to bear on the nature of the mind of man instead of limiting the approach to an arbitrary symbolistic theory. Man should be treated as a dynamic and physiologic whole.

I think that Dr. Harrington's attitude is more a biopsychic approach than a mechanistic approach, and that if he would use that word its meaning and his own altogether lofty advance toward this question would be more easily understood. After all, who would think of trying to discover the meaning of vision without examining the eyeball? If one thinks of sight without reference to the eye, to the

physiology of the retina, to the brain tracts or to the ability of the cortex to gather up and discriminate between sensations that come into it and to unify them, one would know nothing of sight.

DR. GEORGE V. N. DEARBORN: This problem is fundamentally metaphysical or philosophic. It is necessary to get down to realities and to choose between the old medieval dualism started by the church for its own obvious purposes, the dualism that considers body and mind as two disparate entities, and the modern point of view of "spinozistic" monism, which is far, far older than Spinoza. If one can achieve this monistic point of view and thus effectively realize that "mind" and "body" are only aspects of one thing, then one will be more in touch with the modern aspects and trend of psychobiology. One will realize that to talk about purely "psychogenic" disease is utter nonsense. A "mind" wholly separated from a body is inconceivable. I am sure that this is the slow trend of modern psychology and psychiatry. I deem Dr. Harrington's interesting paper thoroughly up-to-date and even not a little ahead of the most common deep-seeing truths along that line.

DR. CLARENCE P. OBERNDORF: A number of years ago, at the Cornell Medical School, when Professor Dana occupied the chair of neurology, he began his lectures with the following statement: "Gentlemen, the textbooks in this course will be three. The first is "Vanity Fair;" the second is "Sherlock Holmes" and the third is Dana's "Text Book of Neurology." Then he added, "You need not bother about the last, but be sure to read the first two." When Dr. Kennedy said that the novelists and dramatists of old were psychiatrists, he said something which has been appreciated for many generations. Dr. Dana wanted his students to learn what was going on in the human mind from the keen delineations of Thackeray and from the acute observations of Sherlock Holmes. When psychoanalysis appeared, with its excursions into unconscious mechanisms, it made advances over ordinary investigations. It has attempted deeper insight into some of the tenets known not only to psychiatrists but to the great authors of the past and present. Here one comes to the question of whether the psychoanalyst needs psychiatry. Of course he does. No one would think of denying that lesions in the brain which are caused by syphilis have a certain effect on mentation. Whatever controls the mind is affected by organic disease in general. In fever, fatigue and hunger there come to the surface some of those interesting phenomena which have been noted by the psychoanalytic school in the unconscious of normal people.

It has been said that the body moves as a whole. It seldom moves synchronously, body and mind together. I doubt whether anybody's mind and body at the present time are moving altogether as a whole. Unconscious registrations are always taking place. The curious manifestations of thought block and depersonalization can be explained in that way—a dissociation of mind and body.

The proposals of Dr. Harrington of right living and education cannot altogether settle the question of mental therapy, because nobody knows what is "right." Furthermore, what is right for one time and place is not right for another. Not that psychoanalysis, as has been proposed here, neglects education, training or environment. In fact, it emphasizes that just these factors have a tremendous influence in the development of desirable habits, especially in early childhood. Moreover, there is no separation of soma and mind in the psychoanalytic tenet. Indeed, the conception is that the body is the seat of vast energy which can invest pathologically any one part of it and prompt certain conditioned impulses. This energy can even so overinvest the mind that thinking itself becomes paralyzed.

When the theory is advanced that by a correct method of living, by altering the environment or by persuasion, which have value, most types of mental dysfunction may be checked, actual practice soon reveals that these remedies do not suffice. One finds that people who have every known blessing in life and in environment, so far as one can determine, who have had everything in their past which would fulfil the requirements of correct education, are nevertheless unhappy and suffering from some form of mental aberration which threatens to destroy them and their families. Psychoanalysis does not neglect in any sense the

impulses coming from the body. Psychoanalytic leaders believe that a practicing psychoanalyst not only should have a good training in general medicine and an extraordinarily good training in psychiatry, but should also have received a thorough instruction in psychoanalysis. Psychoanalysis is trying fundamentally to bring about some correlation between body and mind, and to find the solution for the mysterious manifestations of disease which occur not only in the mind but in the body.

DR. DAVID LEVY: There are certain concepts in Dr. Harrington's paper regarding psychoanalysis and the field of mental hygiene that merit discussion. Dr. Harrington differentiates his own from the psychoanalytic theory and from those of MacDougall and others on the basis of motivism. I do not think that the particular stress he lays on the mechanistic phases of human life really bears out his title. In his paper he himself serves as an example of motivistic behavior. One can see the motive operate throughout his entire paper. He says that it is his intention, or his motive, to explain a concept. He plans to show the weakness of psychoanalytic concepts and the strength of his own particular one. In considering his motives one can accept his hypothesis, as any psychiatrist would, that there are certain inherited influences, some referable to education and some probably also effects of somatic disease, all of which have their bearing on the formulation of his plan and his motive. The psychoanalyst may stress all the factors he stresses and still keep to his own doctrines. They sound to me like the regular formulations which have been used all along in mental hygiene: the study of the individual physically, socially and psychologically. Also, I should say on behalf of psychoanalysis that psychoanalysts have attempted to explain their findings along neurophysiologic lines (Dr. Schilder, for example, before this society), and also by utilizing the conditioned response theory (Dr. Kubie). The finding of physiologic mechanisms does not explain away motive. The child, experimentally conditioned to fear an animal, has a motive when it scampers away. The experiment explains what takes place physiologically in a situation in which the child has a definite objective.

In regard to Dr. Harrington's criticism of the animistic terms in psychoanalytic literature I agree thoroughly. They represent, in many instances, the utilization of analogies for want of more precise definition. Their purpose, it seems to me, is to explain the modifications undergone in instinctive life on the basis of concepts that change with increasing knowledge. In chemistry one spoke of affinities and used similar animistic terms, all of which gave way to increasing knowledge. Likewise, in psychoanalysis the terms super-ego, ego and id, endopsychic censor and the like may be regarded in the future as quaint specimens of historic development.

In regard to heredity, my criticism would be contrary to that of Dr. Harrington, namely, that psychoanalysis does not exclude the importance of heredity but gives it too much stress. Heredity is always the big *X* factor in psychoanalysis. Freud is always stressing the constitutional factor. Why do some children develop sucking problems and others not? Freud thinks it is due to a peculiar innervation of their lips. Why do some people, under a given set of circumstances, become obsessional and others hysterical? The answer given is usually "constitutional factors," by which, for all practical purposes, is meant heredity. The word is used in this sense commonly to fill a gap in knowledge.

In regard to the relation of psychoanalysis to mental hygiene, I think that Dr. Harrington errs in saying that mental hygiene can get nothing from psychoanalysis, since all neurotic behavior is affected by the unconscious which, in turn, cannot be affected by change in environment. It seems to me that it must be clear to any reader of psychoanalytic literature that the unconscious represents psychic material originally affected by the person's social experience. Take Freud's case, for example, of a phobia in a 5 year old boy. What was this boy subjected to? He was exposed to the sight of the aftermath of childbirth. He received a direct threat of castration. Every neurotic conflict represented instinctive reactions to a definite set of circumstances. Hence the knowledge of how the indi-

vidual psychically utilizes social experience throws light on the social situation as an environmental pathogenic factor. As a worker in the field of mental hygiene, I wish to state that psychoanalytic concepts have been utilized as prophylactic measures in just this way. Consider, for example, the primitive hostile feelings of a child toward a nursing baby, material especially elaborated in psychoanalysis, material that gives, in this as in other social relationships, a guide for the prevention or amelioration of situations especially productive of neurosis.

DR. MILTON HARRINGTON: There is only one point in this discussion on which I have anything to add, and that is Dr. Levy's point about motive or desire. Of course, I am not so foolish as to say that there is no such thing as motive: I merely maintain that in attributing an action to a motive one is not getting at its cause. After all, what is meant when one talks about a motive or desire? As I see it, a motive or desire is simply an impulse or tendency to do or seek something. One may say that when a man eats it is because he has a desire for food, or that when he commits murder his motive is revenge, but what does an explanation of this sort really amount to? It amounts to little more than saying that the man does what he does because he has an impulse or tendency so to do, which, of course, is no explanation. In explaining behavior, it is not enough to attribute it to an impulse or tendency; one must also explain the origin of the impulse.

According to the physiologic or mechanistic point of view, the impulses or desires to which people are accustomed to attribute their behavior are merely manifestations of the action of an anatomic mechanism composed of sense organs, nervous system, muscles and glands, and to explain an impulse, therefore, is to explain what happens in the mechanism which gives rise to it. The psychoanalyst, however, looks at the matter in an entirely different way. He ignores the existence of this anatomic mechanism and thinks of a man's manifest impulses and desires as having back of them other deeper impulses and desires which are not apparent, and these deeper impulses and desires as perhaps having still deeper ones back of them. Getting at the bottom of things is for him, therefore, a matter of the disclosure of hidden impulses or motives. A good example of what I mean by this may be taken from Alexander and Staub's book, "The Criminal, The Judge and the Public." In it one is told that many neurotic murders represent disguised suicides, and that when Cain killed Abel he was killing in his brother his own jealousy of his father. It is this practice of ignoring the physiologic basis of desire and looking instead for unconscious or hidden motives which I have in mind when I raise my voice in protest against the motivistic psychology of Freud and plead for a physiologic or mechanistic approach to the problem of mental disorder.

TREATMENT OF PROGRESSIVE EXOPHTHALMOS BY ROENTGEN IRRADIATION.
DR. LEO M. DAVIDOFF.

The exophthalmos associated with hyperthyroidism, although it may remain longer than some of the other symptoms, usually recedes following thyroidectomy. There are a few cases, however, in which the exophthalmos not only fails to recede but may progress after the operation. Interestingly enough, while this progressive, malignant type of exophthalmos is usually an accompaniment of hyperthyroidism, it may occur without any other demonstrable symptoms referable to dysfunction of this gland, but associated in a rather obscure way with cardiovascular-renal disease.

An accurate description of the development of this condition in exophthalmic goiter has recently been given by Naffziger as follows: "Thyroidectomy is performed on a patient with exophthalmic goiter who presents the usual elevated basal metabolic rate and cardiovascular and nervous manifestations. Clinical improvement follows, except that the exophthalmos does not disappear. In a variable period, often in three to four months, it becomes evident that the proptosis is increasing. As it proceeds, an increasing fulness of the lids is noted; then lacrimation and epiphora appear. A watery appearance of the scleral conjunctiva is followed first by edema near the inner canthus and then by swelling which spreads rapidly and protrusion of the inferior palpebral mucosa. Diplopia and lack of

parallelism of the eyes are followed by an increasing limitation of movements of the globes, and downward movements are the only ones retained ultimately. In other directions there may be only slight movement. . . . Such patients are said to be suffering from malignant exophthalmos . . . the severe ones invariably have progressed to the stage of corneal ulceration and infection. Many enucleations have been performed, but the usual termination has been an infected orbit, intracranial extension of the infection and death. . . . Conviction arises that the mechanism in most severe cases is similar in all, whether the patient suffered from hyperthyroidism or not."

While treatment in the mild cases is unessential, except possibly for cosmetic reasons, it is required in the severe cases to prevent serious consequences. Many forms of therapy have been suggested, each one, as a rule, based on the author's conception of the pathologic condition responsible for the disturbance. Thus the orbital fat has often been resected, and the cervical sympathetic ganglia have often been excised, the smooth muscle of the orbit resected and many other types of surgical, chemical and physical methods applied, none of which seem to be effective.

In the past few years, Dr. Naffziger of San Francisco proposed and carried out an operation consisting of a bilateral frontal craniotomy, with removal of the orbital plates and incision of the orbital fascia, for purposes of decompression of the orbits into the cranial cavity. His series of cases must have reached ten or a dozen by this time. In spite of the serious nature of the operation there were no deaths, and improvement occurred in all.

On exposing the contents of the orbit during this operation Dr. Naffziger was struck by the marked hypertrophy of the extra-ocular muscles, and in each case he has removed a fragment of one of them for biopsy. He found, in addition to fragmentation, degeneration and even necrosis, considerable edema of the muscle fibers and round cell infiltration with lymphocytes, young plasma cells and many immature cells of all sorts. This change in the muscle, therefore, he believes to be the cause of the exophthalmos and to account for the marked impairment of rotation of the eyes.

On Feb. 27, 1933, a patient was referred to the surgical service of the Neurological Institute by Dr. Wheeler from the Ophthalmological Institute, for the Naffziger operation. The patient was a Negro, aged about 57, who had been operated on at the Hackensack Hospital, N. J., for hyperthyroidism on Dec. 6, 1930. Previous to thyroidectomy he had had nervousness, loss of weight, "some exophthalmos" and a basal metabolic rate of +45.

The exophthalmos, instead of decreasing, increased steadily after the operation, so that at the time of admission to the Neurological Institute he had tremendous protrusion, especially of the left eye. The conjunctiva of this eye was prolapsed and hemorrhagic, making closure of the eye impossible. Rotation of the eyes was extremely limited, and vision in the left eye was almost nil.

It seemed to me that, in view of Naffziger's description of the pathologic changes in the extra-ocular muscles (especially the round cell infiltration and edema), some hope of improvement of this condition might be expected from irradiation without subjecting the patient to a serious intracranial procedure.

He was, therefore, given a full erythema dose, in divided doses, to the left orbit and its contents between March 4 and 22, 1933. Within ten days improvement in this eye was noted and continued steadily. In August 1933, he returned for follow-up observation, and it was then decided to treat both eyes, but he failed to return after the first few exposures and was lost sight of until about two weeks before he was referred to the Neurological Institute, when it was found that vision had returned to a considerable degree in the treated left (originally the worse) eye, and that vision in the right eye was affected. At the present, he is receiving treatment to both eyes.

The second case is that of a woman, aged 44, in whom this syndrome developed without any other associated symptoms of hyperthyroidism. The exophthalmos appeared in December 1932, after she had been suffering from headaches, dizziness, renal calculi and high blood pressure for a number of years. She complained bitterly of pain behind the eyes, with redness, soreness and a dry feeling of the

eyes. She showed marked bilateral exophthalmos, with inability to rotate the eyes, particularly upward, but no abnormal neurologic signs. In view of the absence of the usual signs of exophthalmic goiter and her inability to rotate the eyes, especially upward, she was suspected of having a tumor of the brain, possibly in the pineal region. Encephalograms, however, gave negative results. Because of the improvement shown by the first patient, she was given a series of roentgen ray treatments to both orbits, and within a month the pain disappeared, the rotation of the eyes improved, the redness of the conjunctivae disappeared and the exophthalmos became less marked.

DIAGNOSIS AND TREATMENT OF SUBDURAL HEMATOMAS: REPORT OF SIXTY-TWO CASES. DR. DONALD MUNRO, Boston (by invitation).

This paper is based on a study of sixty-two cases of subdural hematoma from the Boston City Hospital. All previous studies have been based on series comprising less than eleven cases, with the exception of Jelsma's compiled list of forty-two cases and Coleman's personal experience with twenty cases. The incidence at autopsy, on the other hand, is given as 25 per cent. Clinically, up to 1933, the lesion was diagnosed at the Boston City Hospital in 2.6 per cent of admissions, whereas with the introduction of exploratory subtemporal trephine by me as a method of diagnosis, this incidence rose to 17 per cent.

The experimental work of Gardner in regard to the growth of subdural hematomas is confirmed by the study of the protein content of the fluid clots. Graphs show that the clots liquefy and produce a solution of high protein concentration which induces a flow of diluting cerebrospinal fluid toward the subdural space, dialyzed through the arachnoid; this gradually increases the bulk of the clot and makes it act as an expanding lesion.

The symptomatology, whether considered from the objective or the subjective point of view, cannot be considered as pathognomonic. The same may be said of the cerebrospinal fluid findings. Indeed, I believe that a definite diagnosis can be made only by bilateral exploratory craniotomy. This is indicated, on the one hand, when a patient who has been knocked unconscious following a blow on the head has a headache, mental changes, abnormal physical and neurologic signs, paralysis of one facial nerve and bloody cerebrospinal fluid under increased pressure, and may have a latent period free from symptoms but improvement does not follow proper treatment by dehydration and lumbar puncture, and, on the other hand, when, in a patient known to have a lacerated brain, the neurologic signs fail to clear up and a study of the fluid intake and output relationships and of the cerebrospinal fluid rules out toxic dehydration and meningitis.

As a method of treatment, I recommend subtemporal decompression associated later with lumbar puncture and intravenous injections of hypertonic dextrose solution and, in extreme cases, of 2 per cent solution of magnesium sulphate. Drainage is always provided, and irrigation of a lacerated cortex with physiologic solution of sodium chloride is avoided during operation.

The conclusions are: 1. Subdural hematoma occurs in about one of every six cases of craniocerebral injury. 2. Subdural hematoma may be an expanding lesion. In such cases there may be a latent period during which symptoms are absent until the intracranial pressure begins to rise above normal. 3. The signs and symptoms caused by subdural hematoma are the same as those caused by any other major injury to the brain. Patients suffering from the latter get better under appropriate nonoperative treatment. Those suffering from the former condition, however, in the large majority of cases die unless operated on. 4. The certain presence of a subdural hematoma can be determined only by bilateral exploratory subtemporal trephination. 5. Treatment of subdural hematoma can be best carried out through subtemporal decompression. The entire clot should be removed, drainage should be provided and irrigation in the presence of a contused or lacerated cortex should be avoided. 6. Postoperative edema is a common and serious complication. Treatment should be early and vigorous and include dehydration, drainage by lumbar puncture, the administration of caffeine

and 2 per cent solution of magnesium sulphate intravenously and appropriate measures to lower the body temperature. 7. Without surgical intervention the mortality in twenty-two proved cases of subdural hematoma was 100 per cent. With operative treatment as outlined the mortality in a series of thirty-nine cases was 41.2 per cent. 8. The artificial segregation of certain of the so-called chronic forms of this disease should be abandoned. It has come about only because of failure to recognize the same condition at the time of its original production.

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DISCUSSION

DR. E. JEFFERSON BROWDER (by invitation): I find it difficult to make up my mind as to just what is a subdural hematoma. At the autopsy on a person who has sustained a fatal injury to the head it is exceedingly common to find a contused area of the brain associated with a thin layer of subdural blood. Of course, this small collection of subdural blood more than likely was not the cause of death; it is secondary to a small or large polar laceration. No doubt any blow sufficiently severe to produce such polar laceration would also produce a great deal of general insult to the brain. I have likewise uncovered at operation similar situations and am convinced that it does not make any difference whether such a thin clot of blood is removed or not. It seems to me, therefore, that a subdural hematoma large enough to be of surgical significance is entirely different from a small subdural hematoma such as is found so often at autopsy. In a series of 2,782 cases I found twenty-eight subdural hematomas. Four were chronic; the others were considered as acute. Fourteen of these were discovered at autopsy; the others were found at operation. I am not at all decided as to whether it is best to operate in every case that presents clinical signs of laceration of the brain. If it can be determined that there is a hemorrhage large enough to cause compression, surgical intervention is indicated.

Until one year ago, in cases in which operation was performed the technic was similar to that which Dr. Munro has described. During the past six months, largely because it is so difficult to diagnose a subdural hematoma, encephalography has been resorted to, even in acute cases. I have had twelve such experiences in the last six months and, instead of making the situation worse, in several instances it seemed to me that it was made better by the introduction of air. In three of these twelve cases subdural hematomas were found and operated on that otherwise would not have been diagnosed. I believe that one can inject from 50 to 60 cc. of air with less aftermath than is entailed by an exploratory decompression. It seems rather radical even to suggest the introduction of air in the presence of a lacerated brain, with all the possibilities of secondary hemorrhage; so far, however, no untoward results have been observed.

I have been much interested in Dr. Munro's ideas regarding injuries to the head. I believe that he is a little more radical in his point of view than I have been in the past, although I will admit that I am beginning to operate more in cases of acute injuries to the head than formerly.

As to the chemical explanation of the chronic, or long-standing, subdural hematoma that Dr. Monro has presented, I have no practical experience. His observations are convincing enough, but not conclusive. Dr. Munro's experiences with this large group of subdural hematomas are most instructive. He has shown clearly that ultraconservative, nonoperative treatment when subdural hematoma is suspected should in many instances be supplanted by operative intervention.

DR. THOMAS K. DAVIS: I think that Dr. Munro's subject and the presentation of it are so important that I am sorry he omitted reading part of his manuscript. I should like to have him report on the encephalographic findings, and particularly to describe the mental symptoms in this condition. I think that many patients with lesions of this category have syndromes in which the physical signs are inconspicuous, the mental symptoms predominating. Will he describe the mental symptoms which he might call typical, and particularly their relationship to any latent period?

DR. BYRON STOOKEY: There is one point which is rather confusing. Apparently Dr. Munro has put together two groups of conditions which I have always considered as separate clinical entities; one, the acute subdural hematomas, and the other, the chronic subdural hematomas. It seems to me unfortunate to group these two types, since in my experience they present two different clinical syndromes.

The chronic subdural hematomas seldom present sufficient signs in the beginning to draw attention; most often they follow such apparently slight injuries that the patient himself ignores the accident and a physician is seldom called until much later, when symptoms first appear and the original accident is all but forgotten. Quite the contrary is true for the severe acute cranial injuries in which acute subdural hemorrhages occur. In these the appearance of symptoms is immediate, and there is ample evidence to suggest cranial injury, usually of an overwhelming nature. Thus, the acute and the chronic subdural hematomas are quite distinct, and must be considered as separate clinical entities.

Dr. Munro has stated that most reports have dealt with few cases, seldom over ten, and that unless one has had personal experience with at least twenty cases the material is insufficient to permit conclusions. So few cases have been reported by most surgeons because they have not considered the rarer chronic subdural hematomas with the commoner acute subdural injuries.

Dr. Munro informs me that of the sixty-two subdural hematomas, only nine were chronic types. Thus his experience with the latter is approximately that of most of those who have reported cases of chronic subdural hematoma.

To make the diagnosis of chronic subdural hematoma on neurologic signs alone, without exploration, is often difficult. The signs are confusing; the history of a minor trauma may not be elicited until after the diagnosis has been established. Mental disturbances are at first more or less vague; frequently there are few signs pointing to one side or the other; these are misleading, since not rarely they are on the side of the hematoma. In my earlier experience with these cases I have failed to explore the correct side, but with greater experience I believe a bilateral exploration through a trephine opening should be made.

Dr. Munro has proved in the human being what Gardner has shown experimentally, namely, that osmosis and the increase of fluid in a subdural hematoma are the important factors in the variability of the symptoms. It has always been difficult to understand why these signs appear and disappear, or even why one may have such a long latent period with relatively few signs. In view of Dr. Munro's demonstration, confirming Gardner's conclusions, the variability in the symptoms in chronic subdural hematomas is understandable. As to the operative procedures, I am in agreement with Dr. Munro in preferring to drain in these cases by trephine openings or subtemporal decompressions rather than by an osteoplastic flap. Trotter (1914) suggested small trephine openings rather than an osteoplastic flap. Fleming and Jones, Kenneth McKenzie and others have had good results by these more conservative methods. This has been my own experience, and I am glad to know that Dr. Munro has found this method of value in his large series of acute subdural hematomas, as well as in the nine chronic subdural hematomas included in this series of sixty-two subdural hematomas.

DR. PAUL SCHILDER: In the surgical service of the Psychiatric Division of Bellevue Hospital Dr. Wertheim operated in twelve cases of acute injury to the head. Whether to operate or not depended on the state of consciousness of the patient; deepening unconsciousness indicated operation. Absence or presence of a choked disk and the manometric pressure have no definite value for establishing the indication. Besides the motor phenomena, oculomotor symptoms, such as ptosis, determined the side on which to operate. Pupillary signs were of less importance since many of the patients were alcoholic. Only in one case was no hemorrhage found; autopsy revealed an intracerebral hemorrhage in the area underlying the field of operation, with lacerations and smaller effusions around the place where the skull was opened. It is not always possible to determine whether one is dealing with an extradural or a subdural hematoma. In one case,

with successful operation, an extradural hematoma was unexpectedly disclosed. Our results are comparatively good: Five patients recovered, and seven died; two of the latter had entered the hospital in a grave condition and were operated on only as a last resort.

DR. IRVING J. SANDS: I have had long association with two hospitals, each situated on a congested artery of traffic, and therefore shall discuss the paper in the light of experience gained from the observation of many injuries to the brain seen in these two institutions. It has been my experience that the diagnosis of an acute subdural hematoma, independent of either injury to the brain or laceration of the middle meningeal artery, is extremely uncommon and therefore difficult. The treatment for acute subdural hematoma, if properly diagnosed, should be that suggested by Dr. Stookey in his discussion. Most of the acute subdural hematomas which I found at autopsy accompanied serious injuries to the brain. This naturally leads one to the discussion of such injuries, a subject which is not on the program. Yet it is worth noting that in the treatment for acute injuries to the brain standardization of therapy should be avoided; each patient should receive individual attention. In the publications of Foster Kennedy, Dandy and others there is a tendency toward standardization of therapy. My own experience has taught me to avoid it as much as possible.

DR. DONALD MUNRO: It is stimulating to have such active and argumentative discussion. I think that the subject and treatment of subdural hematoma are a long way from being settled by any work that I have done, but one is not going to get anywhere in any problem in medicine when it is studied from the clinical point of view in series of cases that number less than 20, and usually less than 10. The amount of clinical information to be secured is insufficient and often misleading. The only value I received from any of this work has been from the large number of cases that have passed through my hands.

I have not used encephalography in these cases. I think that Dr. Browder's suggestion is interesting and I am going to try it, because certainly anything that will aid in the early diagnosis of either subdural or extradural hematoma is going to be of great value, if it is not too dangerous, and I think it cannot be too dangerous or Dr. Browder would not be using it.

I do not believe that I am able to consider in detail all the points brought up in the discussion. The mental symptoms are usually those that would lead the family or police to commit a patient to a psychopathic hospital. Some patients have been sent to us by the psychopathic hospitals. One particular patient remained in a psychopathic hospital, with varying diagnoses, including lead encephalopathy. In the more acute cases the patients are apt to be disoriented rather than deeply unconscious, according to my experience.

I did not discuss the disappearance of the clot because I did not dare. I have had thirteen cases in which I have operated and on looking for a clot found nothing but a hole. I have no explanation for it and should not mention it; there had been something recently between the dura and the arachnoid, which had disappeared; a space was found which the brain had not yet expanded to fill. There was subdural fluid there, but whether there had been a previous clot I do not know. I think that it will turn out that a good many of these clots become absorbed, and probably in many cases in which operation would have otherwise been performed it would have been unnecessary because of absorption.

I think that the acute and chronic groups should be kept together. One of the reasons why the diagnosis of chronic subdural hematoma has been so difficult in the past has been because physicians who have seen the patient at the time of injury have failed to appreciate that the patient was not getting well, even though he was up and walking around. They have permitted the patient to go home, and possibly have told him he had concussion of the brain. When symptoms developed and the patient came under the care of another physician the patient failed to mention the previous injury or, if he reported it, minimized it, so that the work has been made more difficult because of the failure of the first physician to recog-

nize the possibility of an acute lesion. I think that these are both parts of one process. The chronic subdural hematomas were once acute, and may have become chronic. That is one point I wish to make; the other is that I believe that operation for traumatic lesions of the brain has now reached a point of which one can afford to say that one is not able to make a diagnosis in all cases, and cannot say whether a fractured skull is present or not, whether an operation must or must not be performed or whether or not there is a concussion or a laceration of the brain. Diagnostic ability is not capable of answering these questions. One must resort to some other process of studying these more mysterious cases, and I believe the safest and most accurate of these methods is exploratory operation. It can be done on both sides in thirty minutes, and it need not involve any decompression unless it is indicated. At the Boston City Hospital my co-workers and I have never failed to find a clot with a brain spoon, if it was present; the medical examiner performs the autopsy in all cases and if there is a clot there he finds it, especially if we have operated. In regard to autopsy statistics, I was brought into this work by the medical examiner, Dr. Timothy Leary, who has collected a large series of subdural hematomas in acute, extensive injuries, which undoubtedly caused death and which were so situated that it was reasonable to suppose that had he been operated on the patient would not have died.

CEREBRAL COMPLICATIONS OF PUTRID PLEUROPULMONARY SUPPURATION. DR. IRA COHEN.

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DISCUSSION

DR. HAROLD NEUHOF (by invitation): May I stress the frequency with which cerebral complications are noted during the course of the type of pulmonary suppuration which Dr. Cohen has described? For example, in the past few weeks there have been four cases of cerebral complications following putrid abscess of the lung at Mount Sinai Hospital. The common occurrence of cerebral complications should therefore arouse real interest on the part of those concerned with cerebral infections. Interestingly, among these four cases are to be seen the different types which Dr. Cohen has described. In one instance a man had had a putrid pulmonary infection ten years before; he was operated on, and a sinus of the wall of the chest persisted. There was no evidence of putrid infection of the lung or of the sinus. Nevertheless, there developed a metastatic putrid abscess of the brain for which an operation was performed recently. Another case illustrates the group of nonsuppurative cerebral foci which Dr. Cohen has termed "aseptic emboli." A brief reference to the case will emphasize the importance of correct diagnosis and the ease with which one can fall into the error of diagnosing a suppurative cerebral metastasis with its probable fatal issue. In this case a chronic putrid abscess of the lung had been operated on a year or more before and a sinus persisted. From this sinus there had been purulent discharge, but more recently hemorrhage from the sinus was the important feature. The patient was admitted to the hospital with fever, unconsciousness and right hemiparesis. The assumption of a suppurative cerebral embolism appeared warranted, but the evidence at hand indicated that the putrid abscess had been adequately cared for. There was active hemorrhage from the wound, and from this an aseptic embolus was thought to have lodged in the brain. The hemiparesis has now disappeared almost entirely, and the present impression is that the patient will undergo spontaneous recovery. It seems to me that careful inquiry as to the status of the pulmonary condition at the time of the cerebral accident is essential in an attempt to make a differentiation between abscess of the brain and an aseptic or relatively aseptic embolus. Dr. Cohen has reported a mortality of 100 per cent in putrid abscess of the brain. I do not think that it is fair to ascribe this mortality solely to the cerebral lesion or to the results of operation, for the active pulmonary focus may be the cause of death. A case too recent to be included in Dr. Cohen's paper

illustrates this point. He operated on a person with putrid abscess of the brain, and the postoperative course, as far as the cerebral complication was concerned, was satisfactory. The operative wound was in good condition; cerebral manifestations had partly disappeared, but the pulmonary lesion was progressive; death may well have been due to the spread of the pulmonary lesion rather than to the cerebral focus.

NEW YORK ACADEMY OF MEDICINE, SECTION OF NEUROLOGY AND PSYCHIATRY AND SECTION OF MEDICINE

C. Burns Craig, M.D., *Secretary*

Joint Meeting, Feb. 13, 1934

Clarence P. Oberndorf, M.D., *Chairman*

Henry James Spencer, M.D., *Chairman*

The Psychogenic Origin of Organic Disease. Dr. Eli Moschcowitz.

1. The following diseases of organic nature are discussed because they are regarded as psychogenic in origin: (*a*) the vast majority of cases of essential hypertension, (*b*) exophthalmic goiter, (*c*) gastric and duodenal ulcer, (*d*) cardiospasm and (*e*) irritable or spastic colon and mucous colitis.

2. In the evaluation of the psychogenic factors, two elements must be considered: first, the underlying personality or constitution, which as a general rule is phenotypic, and second, the exciting factor, which is in the nature of an emotional insult or conflict.

3. There are three stages in the evolution of the organic disease: the constitution, the fixation of an exaggerated function of the organ or organs affected and the development of the lesion.

4. While the personality type varies in these different diseases, there is considerable overlapping in relation to the reaction of the patient to strain and conflict. The emotional crises are not distinctive. Why one organ should be more affected in one person than another is not clear.

5. These maladies are distinguished by five characteristic features: (*a*) From the psychologic point of view they represent exaggerations of normal function. (*b*) They are essentially human diseases and cannot be produced experimentally in lower animals with doubtful exceptions, and then only under conditions that are unphysiologic. (*c*) They rarely occur before the emotive and affective powers are fully developed, i. e., before puberty. (*d*) Their life cycle is characterized by a great tendency to recurrence. (*e*) Their incidence bears a strong relation to world crises or great emotional waves.

6. Psychotherapy is an important element in the treatment of these maladies; obviously its value is largely prophylactic.

Exophthalmic Goiter. Dr. B. S. Oppenheimer and Dr. S. Silver (from the Mount Sinai Hospital).

Certain lesions in the central nervous system are accompanied or followed by clinical features that are considered typical of exophthalmic goiter. The occurrence of acute exophthalmic goiter following severe fright indicates a connection between the cerebral cortex and the thyroid gland, possibly through the thyrotropic hormone of the anterior lobe of the pituitary gland.

Exophthalmic goiter may be associated with other conditions that are generally accepted as being of hypothalamic origin, such as epidemic encephalitis, Parkinson's disease, diabetes insipidus, central obesity, hypogonadalism of the Fröhlich type and central disturbances of temperature regulation. Recently hyperplasia of the thyroid gland has been produced experimentally by the use of the thyrotropic hormone of the anterior lobe of the pituitary and also by the use of cyanide by Marine. The importance of the cervical sympathetic nerve and the insignificance of the thyroid gland in the production of exophthalmos may be noted.

The histories of three cases of typical exophthalmic goiter including exophthalmos, may be presented.

CASE 1.—A girl, aged 18, first presented obesity of the Fröhlich type, with amenorrhea and diabetes insipidus. At this time there were no symptoms referable to the thyroid gland, and the basal metabolic rate was normal. While the patient was under observation there developed tremor, tachycardia, goiter, exophthalmos and a basal metabolic rate of plus 55 per cent. Subtotal thyroidectomy resulted in complete clinical cure.

CASE 2.—A man, aged 40, had been observed at 28 in a typical attack of acute epidemic encephalitis. This was followed by obesity, gonadal atrophy, eunuchoidism and a parkinsonian syndrome. At 39, there began to develop typical exophthalmic goiter with exophthalmos, and he had a basal metabolic rate of plus 41 per cent. Thyroidectomy relieved all the symptoms except the exophthalmos, which has progressed.

CASE 3.—A man, aged 46, presented evidences of diffuse cerebral vascular disease and parkinsonism. He also showed exophthalmos, tremor, tachycardia, goiter and a basal metabolic rate of plus 50 per cent. It was thought that the same vascular process that involved the basal ganglia to produce the parkinsonian syndrome also affected the hypothalamus. This patient did not submit to an operation.

It is possible that some mechanism, working over nerve pathways from the hypothalamus to the anterior lobe of the pituitary gland, may have caused an increased activity of the thyrotropic hormone and resulted in hyperplasia of the thyroid gland in this group of cases.

THREE PSYCHOSOMATIC SYMPTOM PHASES WITH AN EMOTIONAL COMMON DENOMINATOR. DR. GEORGE DRAPER (from the Presbyterian Hospital).

An American professor, aged 36, presented three major symptom phases which involved three so-called main systems of the total organism—digestive, circulatory and ideational. The symptoms in each of these systems were the expression of an emotional conflict which had its origin in the Oedipus situation. The patterns for the symptoms in each system were definitely laid down in infantile experience. Abdominal cramps, high blood pressure and the fear of homicidal attacks have all yielded to psychoanalytic treatment.

PSYCHOGENIC FACTORS IN EXOPHTHALMIC GOITER. DR. GEORGE W. HENRY (from the New York Hospital).

Four cases illustrating various aspects of psychogenesis in exophthalmic goiter are presented. The first patient, a married woman, aged 30, came to the hospital in October 1932, with the complaint of an enlarged thyroid gland. She had noticed a swelling for ten years. She had difficulty in swallowing and hoarseness and was inclined to be nervous. The symptoms increased following childbirth in September 1929, and again in April 1932. She had taken iodine intermittently since the first child was born. The basal metabolic rate was plus 68, and the condition was regarded as typical exophthalmic goiter; operation was advised. The patient could not leave her work, however. A month later, she was persuaded to place the second child in an institution. She had worried much and lost sleep over the child, who was hydrocephalic and mentally deficient. In February the

child died. Two weeks later the patient's basal metabolic rate was plus 25. She gradually improved until a year after admission she was practically free from symptoms; the thyroid was firm and much smaller, and the basal metabolic rate was minus 11. The internist regarded the result as phenomenal, especially since she had had no therapy except the aid in readjustment to the illness and death of her child.

The second patient, a woman, aged 28, had adjusted herself poorly to economic problems and to marriage. Throughout childhood she had pictured herself as a popular, beautiful and admired woman. In 1926, in her first year at college, she was forced to earn her own living after having been comfortably situated. During the next two years she had three love affairs and one abortion. In 1928, she married, but soon had trouble with her husband's family. She felt bored at home and went to work. Two years later a subtotal thyroidectomy was performed, following a not unusual history of exophthalmic goiter with a basal metabolic rate of plus 55. After another two years the symptoms returned. She was advised to stop work and tried to convince herself that taking care of a home was an interesting job. She began to doubt whether she was really in love with her husband. She refused to accompany him on a trip to visit his mother, and a few days before he had planned to go alone she attempted suicide by taking phenobarbital. After a period of five months of psychotherapy in the psychiatric clinic she has fully recovered from the exophthalmic goiter and feels that she can meet her life situations and that she is more stable than she has ever been.

The third patient experienced unusual emotional stress from childhood. She was 44 years of age and married. Some of her problems are best described in her own words: "My chief trouble was with my mother. She was irritable and found fault with everything. I would say nothing but cry. She picked on me because I was the oldest. I was broken-hearted because she thought more of the other two children, although I was earning more. She would always tell father and he would beat the life out of me. I have no recollection of him except for the beatings he gave me. He died when I was a little girl, and mother always hated me. She didn't want to have children. I had little schooling because my mother was insistent that I stay home and help with the housework. I was very unhappy—the unhappiest girl that ever walked. I got married to get rid of her, and then I had to take her with me for five months before she died." She was married at 27 to a widower, aged 47. The husband has always been kind to her, but she has never felt any affection for him. She was disappointed in not having been pregnant. A few years before marriage she gave up a young man on the insistence of her mother.—"He had the ring bought. I am really Mrs. D——. I'm living in the past with the other man. He never got married. You have only one love in your life and that was torn away from me and I'll never have another. That was twenty-two years ago. I don't know where he is."

Before the onset of the symptoms of thyroidism in 1927, she was depressed and anxious. "Previously I was happy-go-lucky, but for six months my husband said I acted like an insane person. When I got melancholy, I thought I would go to Kings Park like my grandmother. It was terrible the way that would prey on my mind. I used to try to dope out the easiest way to do away with myself. For two years after the second operation I felt like a new woman. Then they brought my husband home ice-cold in an attack of angina (February 1930). The doctor said he wouldn't live through the night. Then I started to get all my symptoms back again. I was melancholy, lost weight, was sleepless, cried all the time, thought everybody hated me. I was quite apprehensive that my husband would have another attack. If he dies, I am alone."

Around Christmas, 1932, she was short of breath on exertion. "I started to get melancholy again. I felt like crying all the time. Before I came here I didn't care whether I lived an hour. My sister said, 'You must like to be sick.' The minister said my trouble was imaginary. He might just as well have stuck a knife into me. My sister is like my mother—she has feeling only for herself.

If I have an operation, I don't care whether I get well or not. My brother sneers at me. He says it's all imagination. I'm heart-broken." On returning home from the hospital she learned that her nephews had been planning the disposal of her property, because they had expected she would die. "It was terrible when I got home. I screamed. I said 'Take me anywhere—Kings Park—anywhere to get out of here.'"

In recent years financial troubles have been a great problem to her, and since leaving the hospital she has been trying to support herself and her husband by sewing. Her relatives on her mother's side of the family are well situated financially, but "they made fun of me when I married because my husband was so old. I never see any of them. They are not relatives but enemies."

The fourth case is somewhat spectacular. The patient was an unmarried student, 25 years of age. She came to the hospital because of a recurrence of symptoms of hyperthyroidism and a swelling of the gland of four months' duration. She had adhered to the orthodox Hebrew teachings of her childhood and had assumed that the proposals of intimacies by young men were one of the unavoidable and unpleasant features of enjoying their company. She felt extremely confident of herself and met those proposals with ridicule. Two engagements were terminated within a few weeks because "I never particularly cared anyway. If they cared I did not." After keeping company for three years with a third man, ten years her senior, she was assaulted by him while she was intoxicated. For weeks thereafter she went about as though dazed, frequently weeping, but she never confided with any one regarding the cause of the change in her behavior until she consulted me four years later. About a week after the assault the man proposed that relations should be continued since she was no longer a virgin. She then commanded him to leave, but as he did so she realized that she was in love with him. Within a month after this parting the classic picture of exophthalmic goiter with marked exophthalmos developed. A thyroidectomy was performed six months later, but there was little subsequent improvement in the exophthalmos. Two years after this operation all of the symptoms returned, and another subtotal thyroidectomy was performed. It was because the surgeons gave her little encouragement as to a permanent recovery that she appealed to a psychiatrist for help.

It was soon learned that the psychic trauma already mentioned was only the beginning of an indefinite period of stress and unhappiness. She was unable to give up the man, and yet his attitude was such that she could not accept his attentions. He criticized everything she said, did or wore, and he continued to make sexual proposals. As regularly as he did they would quarrel and separate. In a few weeks she would again feel anxious to see him, and they would meet but only to quarrel again. She now thinks that their relationship is continued merely through habit, that there is no purpose in going on and that she really hates him. Yet she cannot free herself from him.

She has lost confidence in herself, avoids people because of the marked exophthalmos and feels that there is no solution for her problems. Her family were once in comfortable circumstances and she was self-supporting. Now they are in need and she is an additional burden to them. "It was a sort of a game that turned into reality. I tell you this because I see no way out. Perhaps you can help me."

DISCUSSION

DR. FOSTER KENNEDY: I believe that the mind can cause functional disease, and that functional disease long continued can result in organic change. Many might admit the possibility of crucifixion stigmas in a hysterical person, but that does not gainsay the fact that urticaria is more commonly an allergic disease than a disease of psychogenic origin.

Both the organicist and the advocate of the psychogenic idea are led to extremes by the fact that they are "appointed," as it were, by their fellows on one side or the other. They are given a standard to carry. If they are wise, they will know that both standards cover truth. I think myself that function is implicit in struc-

ture, but one lives in a world of which one knows very little, and it may be that vibrations are more important than matter and that matter may be the expression of energy. However, I can think better in terms of matter than I can in terms of energy, and I find it more possible to be explicit to myself in the former idiom; all this, however, is but personal technic!

DR. REUBEN OTTENBERG: I feel a sense of insecurity when I discuss a subject which is not susceptible to methods of laboratory precision. The psychogenic origin of organic disease has not yet been subjected to laboratory criteria. The thesis that Dr. Moschcowitz presented has not yet been subjected to measured demonstration. As Dr. Kennedy pointed out, in considering a subject of this kind men are likely to take sides as a matter of opinion or even to be appointed to take sides. I may offer myself as a demonstration specimen; I am today a recent convert, who comes to this meeting, gives his personal reactions and declares himself converted. My initial reaction was conditioned by my training and I was against the idea that organic disease can be due to psychogenic causes. Twenty-five years ago, when I went into practice, I should have derided the idea that organic disease can have psychogenic causation. It has been only after experience in medical practice that the conviction has been forced on me, in spite of my skepticism, that this can occur. I cannot take the time to offer detailed evidence, but I have seen many instances that seem impossible to explain in any other way. When I went into practice many doctors wore beards. Today beards have gone out of fashion. It was said at that time that there were two kinds of doctors: those with beards, the human kind, who went into the innermost aspects of their patient's lives, understood them and cured them, and the smooth-shaven doctor who made measurements, was a "hard-boiled" scientist and would believe only those things that he could detect by means of his five senses. I was one of these. I shaved and had no use for a beard. But twenty-five years of experience has forced the conviction on me that organic disease can have in some instances a psychogenic origin. Since coming to this conclusion I have been debating with myself whether it would not be wise to raise a beard in keeping with my changed opinion.

The questions arise: What is meant by organic disease? What is meant by functional disease? Where does functional disease stop and organic disease begin? There are many conditions difficult to call either purely functional or purely organic, for they merge so gradually into one another. Functional disturbances come first and organic changes follow. Asthma is primarily due to spasm of the bronchial musculature; definite morphologic changes follow. Does asthma begin as a functional disease and become organic? These are questions that require careful study and critical analysis. I never believed that asthma could have a purely psychogenic basis until two or three years ago when Dr. Oberndorf showed me a case of typical and severe asthma. The patient was in status asthmaticus. She received anti-asthmatic therapy, a dozen hypodermic injections of epinephrine a day and morphine constantly. Nothing of a pharmaceutic nature had done any good. The patient was sensitive to a few proteins and was immunized to them, but the asthma continued unabated. Finally, perhaps in desperation, a purely psychologic approach was attempted and she made a complete recovery. If I had not seen this myself I would not have believed it. This, I believe, was a real case in point.

There are many other instances in which it is difficult to say where functional changes end and organic changes begin. Changes in blood pressure and in blood sugar have already been mentioned. It was reported recently from a foreign clinic, detailed measurements being given with scientific basis of proof on a large scale, that a rise of bilirubin in the blood can occur from psychologic stress. The evidence was clear and concise and left no doubt as to its authenticity. This does not ordinarily progress to clinical jaundice, but the rise of bilirubin in the blood is the beginning. One is reminded of the old phrase "bile" as representing anger. Inhibition of secretion of bilirubin by the liver may therefore be a functional disturbance. The opposite kind of functional disturbance, oversecretion rather than suppression, that of hydrochloric acid in the stomach, is, I believe, a predisposing condition to peptic ulcer.

Personally, I am convinced that there are many cases of chronic colitis, so-called "nonspecific ulcerative colitis," that have a psychogenic origin. The streptococcus isolated by Bargen has not yet been confirmed as the etiologic agent. It may play a purely secondary rôle. Investigators are now looking for, and they think they are finding, a psychogenic etiologic causation for exophthalmic goiter. Attention is being turned to the possibility of a psychogenic etiology in nonspecific diarrhea. In the presence of constitutional factors such as Dr. Draper has studied so thoroughly the psychic insult appears to function as a trigger and set off the organic disease. It is, of course, undoubted that many types of organic disease cannot be produced in this way.

Studies in recent years in the hypothalamic centers indicate that they are under the control of the cortex of the brain and that they in turn control many vegetative functions. That suggests a possible explanation of the fact known to all clinicians and taught me in medical school that diabetes often follows psychologic trauma. Parkinson's disease is stated sometimes to follow psychic trauma. I do not say that the lesions that occur in that disease are due to psychic trauma primarily, but it may act as a trigger to set off functional disturbances which in turn may eventually lead to organic lesions. Diabetes insipidus has also been attributed often to psychic factors.

It seems to me that there are many situations in which there is already a beginning organic disease or a predisposition to it when a psychogenic cause acts as the necessary inciting agent. For example, in angina pectoris the heart may be previously damaged and give no clinical attacks; they are brought on by some psychogenic trauma. The background of the disease is in the coronary arteries, but the actual attacks may be brought on by the sudden psychologic load, and the attacks may in turn lead to further damage of the cardiac muscle.

On the subject of infection, tuberculosis or influenza is certainly not due to psychogenic causes, but those who went through the influenza epidemic in 1919 saw that people who were worried, who worked hard and who had many burdens to carry usually succumbed with the most severe form of the disease. The mentally burdened student type is particularly susceptible to tuberculosis. It appears as if psychogenic causes without directly producing organic disease can act as predisposing factors.

One must not be misled. As a clinician one must not go too far; it is healthy to cloak enthusiasm in a mantle of open-minded skepticism. How can one prove that a case is of psychogenic origin? One must first exclude organic etiology, and only then is one warranted in seeking psychogenic complications. I have in mind two patients who were in the same ward about one month ago. Both apparently gave the same complaints and presented the same clinical picture, one that was mentioned here. I refer to cardiospasm. Cardiospasm is being found to be due frequently to psychogenic causes. These two patients were of about the same age. Both of them had many psychogenic factors and both were interviewed by the psychologist of the institution. The one who had the least psychogenic factors turned out to have true cardiospasm. My co-workers and I were convinced for a long time that the other patient also had true cardiospasm; we were fortified in that opinion when we elicited a most harrowing personal history. Roentgen examination seemed at first to confirm the diagnosis. It was later found that it was impossible to get the usual weighted mercury tube past the spastic cardiac muscle at the entrance of the stomach. Finally esophagoscopy was done; a biopsy specimen was removed, and a diagnosis was made of carcinoma of the lower end of the esophagus. This emphasizes that the diagnosis of psychogenic causation can be made only when organic etiology is definitely excluded.

I am personally convinced of the psychogenic origin of certain organic diseases, yet I feel that I must be wary and have left it in my mind as much of an open question. A great deal more evidence is needed, such detailed evidence as Dr. Henry presented, before one can accept the thesis as scientifically proved.

DR. ROBERT B. MCGRAW: There seems to be no need, although I thought there would be at the start, to "sell" the idea that psychic causes produce physio-

logic changes and later structural changes, but there is need still to discriminate in respect to the approach in treatment; whether the condition should be treated by medicaments and regimen or by psychotherapy; whether, in other words, the psychotherapy should proceed on traditional medical lines or should proceed along thoroughly psychologic lines. Fear and chronic anxiety do not seem to be very different, after all, but as to the cause of the chronic anxiety and as to the specificity of the location of the lesions there is room for a great deal of controversy. Why one person at one time breaks in respect to the cardiac function and another in respect to the gastric, the thyroid or the colonic function is worthy of some speculation, some observation and considerable investigation, both clinically and experimentally. I do not think that any one here believes, for example, that oral or gastric fixation or rapid living is the only answer.

Sometimes superficial treatment has to be chosen to the exclusion of possibly more pervasive (deeper and more inclusive) methods. Dr. Moschcowitz has wisely noted the so-called spontaneous cures. They are called spontaneous because it was not known why the patients recovered. Dr. Henry indicated, in some of the cases with what might be called natural histories, i. e., histories not much influenced by medication or psychotherapy, why he thinks the patient got well. Possibly some of the patients who recovered spontaneously from exophthalmic goiter recovered because they matured psychically or, better, psychosexually.

If these patients are to be treated by any thoroughgoing system or method of psychotherapy, it seems to me that the medical profession is entitled to know the course subsequently. This condition has been most admirably met by Dr. Draper. He has shown a case which has demonstrated defects in several systems, and he has given some hints as to why these different systems are affected. He suggests that the psychic systems are the most fundamental. Will he tell whether he has changed his views in respect to constitution?

Dr. Carl Binger: Regarding Dr. Ottenberg's comments on the importance of making measurements there can, of course, be no doubt, but obviously one must observe before he can measure. The fact that the subjects under discussion do not lend themselves to measurement does not, I think, detract from their importance. I was much impressed with Dr. Moschcowitz' presentation and with the accuracy of his observations. The recent work of Dr. Alexander, not yet published, in some measure corroborates Dr. Moschcowitz' findings as to the type of persons in whom peptic ulcer is prone to develop. Alexander and his collaborators have studied a group of patients suffering from peptic ulcer by the psychoanalytic method. He believes that he has established an emotional situation common to this group and has defined a recognizable type, though to be sure it may not be a specific one.

Dr. Moschcowitz emphasized the feature of hyperfunction characteristic of those pathologic states in which the emotional situation was, in his opinion, of paramount importance. I believe that this is true, but that one may also find important emotional conflicts in pathologic states other than those represented by so-called hyperfunction. I have in mind conditions in which the element of hyperfunction is less readily discernible than it is in hypertension and peptic ulcer. For example, I have under observation a young woman who has at various periods of her life, beginning with early childhood, suffered from corneal ulcers. She is of the hysterical type. When one sees her first, one notices her wide open eyes. Indeed she seldom appears to wink. Whether this fact has a bearing on the corneal ulcers, I do not know. She has also had, according to the ophthalmologist who has taken care of her, a vascular lesion of the retina of mysterious origin. Accompanying these two pathologic changes there has been, since early childhood, a tremendous scoptophilic interest. She has always wanted to see, and seeing has played one of the leading rôles in her whole emotional development. Under psychoanalytic handling, treating her entirely from the side of her emotional conflicts, not only have the lesions of the cornea vanished but also the vascular changes in the retina, according to the statement of the ophthalmologist.

DR. E. D. FRIEDMAN: If Dr. Moschcowitz would be willing to modify his title and to say that psychogenic factors may aggravate existing disease or precipitate disturbances in the endocrine and involuntary nervous system, one would be inclined to be more whole-heartedly with him in his concept.

Neusser of Vienna, used to speak of emotional glycosuria and "examination" diarrhea in students as expressions of disturbances in the endocrine and involuntary nervous system resulting from psychic trauma. Recently, Erdheim, of Vienna, was holding a lecture and happened to show pathologic material from a case of coronary thrombosis. An American student in the audience commented on the fact that in the relatively large postmortem material which Erdheim has at his disposal cases of coronary thrombosis were relatively few in number. Erdheim's reply was, "Have you ever seen a Viennese in a hurry?" But hurry alone is not the cause of coronary thrombosis. Those persons in whom physical exertion or psychic trauma evokes a coronary attack must have had preexisting disease of the coronary vessels. The psychic trauma probably serves as a precipitating factor by adding the element of vascular spasm to an already existing arteriosclerotic process.

In many of these cases there probably is a constitutional factor. Dr. Moschcowitz has already hinted at the importance of constitution in atherosclerosis, hyperthyroidism and peptic ulcer. In hyperthyroidism, as he has already stated, status lymphaticus is a frequent coexisting phenomenon. The importance of the disturbances in the involuntary nervous system in hyperthyroidism was known to the older clinicians. Charcot and Oppenheim looked on hyperthyroidism as a sympathetic neurosis. The frequency with which essential hypertension occurs in families also seems to indicate a constitutional trend toward vascular disease. Dr. Moschcowitz has already referred to the spasmogenic theory of peptic ulcer. Ischemic necrosis due to repeated vascular spasm may serve as the basis for the subsequent development of ulcer.

It seems to me also that the division of disease into functional and organic should be abolished once for all. Soma and psyche are intimately interwoven. Disease expresses itself first in disturbances of function (pathologic physiology). When these disturbances in function have continued for some time they eventuate in the pathologic-anatomic changes which are witnessed on the autopsy table.

DR. ELI MOSCHCOWITZ: I was particularly impressed with Dr. Ottenberg's remarks. A study of the psychogenic aspects of disease is apt to lead to an overemphasis of their importance. One must, as Dr. Ottenberg said, exclude an organic cause of disease first, and then one has a right to think of a possible psychogenic origin. No one realizes better than I do the deficiencies of this paper. I have merely touched on a subject that has vast implications. There is a tremendous amount of work to be done, not only in the psychogenic approach but in the interpretation of the mechanisms. The elucidation of the mechanism, after all, is an important job. My real purpose in presenting this paper was to try, if possible, to stimulate a more vitalistic approach to the study of disease. The mechanistic approach has been too dominant.

Book Reviews

Psychoanalysis of the Neuroses. By Dr. Helene Deutsch. Price, $4. Pp. 230. London: The Hogarth Press, 1932.

This book is in its way almost a masterpiece of descriptive clinical pathology. In medicine the underlying disorder must be demonstrated at the bedside, but in psychiatry, and particularly in psychoanalysis, the only method available is the study of case histories. This book consists of a series of case histories which are used to demonstrate psychoanalytic theories. The author belongs to the orthodox freudian school, and everything which she writes is orthodox and based on analytic experience. The theoretical part of the book takes its point of departure from Freud's more recent works, particularly "Beyond the Pleasure Principle," "The Ego and the Id" and "Inhibition, Symptom and Anxiety." It presupposes some basic knowledge of psychoanalysis and its terminology. The theoretical discussions are based on Freud's more recent theory of the diffusion of the instincts, and the case histories form, as it were, a chronological series which illustrate the working of these forces. The book is not a textbook or a compendium of cases, but resembles rather a show case of exquisitely mounted specimens in a museum of clinical pathology. All the superfluous débris is cut away, and one may see at a glance the connecting links in the system. Because of its lucidity of thought, simplification of theory and graphic style, the book is peculiarly adapted to students of psychoanalysis. No doubt it will also appeal to many psychiatrists who are sympathetically inclined.

The introductory chapter deals with the three etiologic factors concerned in the neuroses: the frustration, or the external precipitating factor, regression and fixation. The author shows how the actual cause in the formation of a neurosis is never specific. It merely creates, or reactivates, an internal conflict which is impossible of solution because there are difficulties in the personality, and consequently symptoms appear. External frustrations are repeatedly happening in the outer world. The healthy person is able to find some way out of these dilemmas; either he attempts to change the external situation or else he renounces the object of his desires and seeks gratification elsewhere. The neurotic person is unable to do this because of an inner indecision. This question of the actual conflict and the internal conflict has a further importance in therapeutic procedure. In recent writings Rank stated in criticism of analytic treatment that the actual situation is often neglected to the benefit of the historical material—that is to say, the patient's infantile traumatic experiences. Helene Deutsch shows by an example that the actual cause or present situation of the patient can be understood only in terms of the past. It is equally true that the past can best be interpreted in terms of the present. It often appears that the external situation is merely an *agent provocateur*. At times patients themselves provoke the precipitating factor in order that they may live out neurotic tendencies of which they are unconscious.

In a case described, an original traumatic experience of childhood which produced the neurosis that led to the disturbances in adult life was itself an external frustration in childhood, and produced ill effects only because of the disposition of the child. Thus a continual series of links is set up between the present and the past. Both the present existing situation and the internal neurotic conflict, must be taken into account; the one can be understood only in terms of the other.

The example mentioned illustrates the author's ability to make clear an intricate problem. It throws light also on many problems which have been only imperfectly understood: the relation of the actual conflict to the so-called traumatic injuries of childhood, the mechanism of pavor nocturnus, the importance of the companion in agoraphobia, the possible etiology of ticklish zones and the relationship of agoraphobia to hysteria, on the one hand, and to obsessional neuroses on the other. There are many useful hints in regard to analytic practice, although as a general

rule the author confines herself to clinical descriptions. The great value of the book is its representation in graphic form of the psychoanalytic concept of the neuroses. Perhaps at times the author has simplified her material too much; She is perhaps too prone to make statements that would carry more weight if there were further historical material. For instance, in describing and explaining hysterical convulsions, she states merely that analysis has proved them to be a motor dramatization of certain fantasies representing coitus, rage, birth, etc.; although there is no reason to doubt this, one would like to know more about the analytic material which led to these conclusions.

The translation, on the whole, is good, although there are one or two expressions that might be altered. For instance, "actual" is usually taken over literally from the German, where it has an entirely different meaning, which perhaps might better be translated as "current." Again *Entmischung* is translated as "decomposition," whereas the word "diffusion" would seem more fitting. The book is to be highly recommended.

Inanition und Epilepsie. By Carl Clemmensen. From Acta psychiatrica et neurologica supplementum III. Copenhagen: Levin & Munksgaards, 1932. Price, 8 Danish crowns. Pp. 164.

This study, based on starvation treatment of one hundred and fifty-five epileptic patients, is a lengthy and unusually detailed investigation into the relationship between starvation therapy and epileptic seizures of all types. From four to five days of fasting brings about cessation of the seizures with great constancy. The duration of the disease has no bearing on the results of fasting; all the epileptic manifestations are equally influenced, and both "organic" and "cryptogenic" epilepsy are influenced. The cases most suitable for starvation therapy are those in which the seizures are very frequent. A great deal of laboratory work was done on these patients, but no new facts were brought out.

The author suggests that the cessation of seizures during starvation is secondary to the dehydration accompanying starvation. He does not definitely commit himself in regard to either etiology or specific therapy, but states that the starvation-dehydration-hydrogen ion concept forms an acceptable working hypothesis. His work is detailed, and his conclusions are broad. No new concepts are advanced, nor is any original work presented. Many previous investigations have been repeated. His emphasis on dehydration as the primary therapeutic factor in starvation is not well substantiated by his investigation.

The working hypothesis advanced is as follows: "When the organism, under fasting conditions, has to burn fats and at the same time, has only a small quantity of carbohydrates available, the combustion of fats results in the production of a large quantity of acetone bodies and sometimes a severe acetonemia develops. This acetonemia brings about a displacement of the acid-base equilibrium towards the acid side. Together with this shift of acid-base, and partially because of it, there develops a state of dehydration in the organism. The dehydration leads to a cessation of seizures. How it does so is not known. According to Temple Fay, it is probably because of the diminution of the subarachnoidal oedema over the fronto-parietal areas of the brain. The role of the circulatory system in the effect of dehydration on seizures is not clear; it is possible, however, that a slight reduction in blood vessel tonus may be of some significance."

The author's hypothesis is open to criticism, because it has never been conclusively shown that the cessation of seizures is a direct result of dehydration per se, because the pathologic concept advanced by Temple Fay is untenable and because the rôle of vascular tonus in dehydration is too hypothetic.

Peripheral Nerve Injuries. By Lewis J. Pollock and Loyal Davis. Price, $10. Pp. 678. New York: Paul B. Hoeber, Inc., 1933.

The combination of a first-rate neurologist and neurosurgeon is necessary to produce such an excellent book. Neither could have done so well alone. The

material gathered during the World War has been used as the basis of occasional critical articles on various aspects of the problem, but this book represents the critical opinion of the authors after mature deliberation. It begins with an excellent presentation of the methods of examination, and here the authors show considerable ingenuity in some of the methods of testing. Unless such methods are accurate and painstaking and are done with instruments which are fairly dependable, conclusions cannot be depended on. There is nothing more difficult and more time-consuming than the measurement of motor loss, to say nothing of the estimation of sensory disturbances, which depends, in the main, on accurate instrumentation and cooperation of the patient and the examiner as well.

In the estimation of motor loss, the authors properly stressed the so-called supplementary, or trick, movements, that is, those not dependent on the motor lesions. They considered the impairment of movements due to gravity and changes in the joints. They employed a spring scale for the measurement of power, an ingenious tonometer for the measurement of tone, loss of water displacement for the measure of the degree of atrophy and graphic methods for the measurement of atrophy, loss of striation of the skin, etc.

Their methods of sensory examination were good enough for the period and the best available in war times. The authors differ with Head and agree with Stopford as regards the interpretation of the epicritic sense, and insist that in making an estimation of the return of sensation it is necessary always to determine the isolated supply of the nerve before accurately judging its meaning.

The book is divided into thirty-seven chapters. After a discussion of general clinical symptomatology, embryology, pathology and nerve impairment, the surgical aspect is emphasized. Each nerve is discussed from all standpoints. Finally, there is a chapter on cranial nerves and the results of peripheral nerve surgery. The statistics are surprising. There are 312 excellent illustrations and a complete bibliography. The publisher has done his work well. Occasionally Weir Mitchell is spelt Wier Mitchell, but with such minor exceptions this is undoubtedly the best book on peripheral nerve injuries that has appeared since the classic work of Weir Mitchell, and no higher praise can be given it than to class it as a worthy successor.

Les traitements modernes de la paralysie générale. By A. Sézary and A. Barbe. From La pratique médicale illustrée. Price, 8 francs. Pp. 35. Paris: Gaston Doin & Cie, 1930.

This is a short monograph on the modern treatment of dementia paralytica. The authors call attention to the inefficacy of the usual antisyphilitic therapy (mercury, bismuth and the arsphenamines). They give their results with the use of the sodium salt of acetarsone (acetylaminohydroxy-phenylarsonic acid) and compare these results with the results of malaria therapy as recorded in the literature.

The authors divide the hundred and twenty-five cases in which acetarsone therapy was administered into three groups: the expansive cases (thirty-one), those in which there was slight mental deterioration (forty-one) and those in which there was dementia (fifty-three). In the expansive group they obtained good results in 68 and poor results in 32 per cent; in the mildly demented group, good results in 58.5 and poor results in 41.5 per cent, and in the demented group, good results in 26.4 and poor results in 73.6 per cent. The serologic results with the cerebrospinal fluid were: reduction of the Wassermann reaction in 34 per cent, decrease in the pleocytosis in 73 per cent and decrease in protein content in 47 per cent.

The authors summarize the literature on the results of malaria therapy in dementia paralytica and state that the results that they obtained with acetarsone compare favorably with the average results of malaria therapy. They point out that there are practically no contraindications to the use of acetarsone, whereas malaria cannot be used in many cases. They also emphasize that malarial therapy is always accompanied by a definite mortality, whereas there is no mortality with acetarsone. They believe that with acetarsone and malaria therapy they have

"made great strides" in the treatment of dementia paralytica, but that they "have not completely conquered the disease" and further research is necessary.

Habits, Their Making and Unmaking. By Knight Dunlap. Price, $3. Pp. 322. New York: Horace Liveright, Inc., 1932.

The first chapters of the book deal with the problems and principles of learning and lead up to the author's interesting theory on the subject. He believes that for many types of response, repetition does not increase the probability of the occurrence of the response on presentation of the stimulus, but decreases it. The repetition of a bad habit under suitable conditions will break the habit. The specific habits discussed are stammering, tics and bad sexual habits. From the author's exposition here it is clear that the "negative method" of habit breaking is not the only factor in his treatment. The stammerer, for instance, must understand the nature of his defect and desire to speak fluently; he probably receives a large measure of explanation, encouragement and suggestion, all of which help him in his readjustment. Detailed analyses of cases of bad habits in the patients treated by the "negative method" are of great importance in evaluating the theory, and it is to be hoped that the author will publish some of his studies. The present book is essentially a popular treatment and one which Dr. Dunlap apparently wants in the hands of the general public, in spite of the fact that he believes that his method of habit breaking should be attempted only by persons of wide experience and psychologic training.

Adjustment and Mastery. By Robert S. Woodworth. Price, $1. Pp. 137. Baltimore: Williams and Wilkins Company, 1933.

It would be difficult to imagine a more excellent simple approach to the general science of psychology than that presented by Professor Woodworth in this small book of less than 150 pages. The emphasis is placed on problems of adjustment to environmental conditions and mastery of them, and the progress which psychology has made toward the solution of these problems is set forth briefly and clearly. Some of the difficult problems still far from solution are indicated, but the author has written for the layman rather than for the specialist. The book will undoubtedly be successful in interesting the general reader and in giving him some idea of the purposes of psychology and of its relation to his individual problems.